“PREVALENCE OF HYPOMAGNESEMIA & HYPOKALEMIA IN PATIENTS WITH STEMI & ITS RELATIONSHIP WITH OCCURRENCE OF ARRHYTHMIAS”

A DISSERTATION SUBMITTED TO THE TAMILNADU Dr. MGR MEDICAL UNIVERSITY

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In partial fulfilment of the Regulations

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M.D. (GENERAL MEDICINE) BRANCH-I

DEPARTMENT OF GENERAL MEDICINE

TIRUNELVELI MEDICAL COLLEGE

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CONTENTS

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CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC

REF NO:856/GM/2016

PROTOCOL TITLE: PREVALENCE OF HYPOMAGNESEMIA & HYPOKALEMIA IN PATIENTS WITH STEMI & ITS RELATIONSHIP WITH OCCURRENCE OF ARRHYTHMIAS.

PRINCIPAL INVESTIGATOR: Dr. D.DEVANATH, MBBS.,

DESIGNATION OF PRINCIPAL INVESTIGATOR: POST GRADUATE IN GENERAL MEDICINE

DEPARTMENT & INSTITUTION: TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI

Dear , Dr.D.Devanath, MBBS.,, The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 05.08.2016.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of the Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At the time of PI's retirement/leaving the institute, the study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
   d. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IEC, only then can they be implemented.
   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g. Any deviation/violation/waiver in the protocol must be informed.

STANDS APPROVED UNDER SEAL

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CERTIFICATE - II

This is certify that this dissertation work title **"PREVALENCE OF HYPOMAGNESEMIA & HYPOKALEMIA IN PATIENTS WITH STEMI & ITS RELATIONSHIP WITH OCCURRENCE OF ARRHYTHMIAS"** of the candidate **Dr.DEVANATH.D** with registration Number **201511352** for the award of M.D. in the branch of **GENERAL MEDICINE.** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **1 percentage** of plagiarism in the dissertation.

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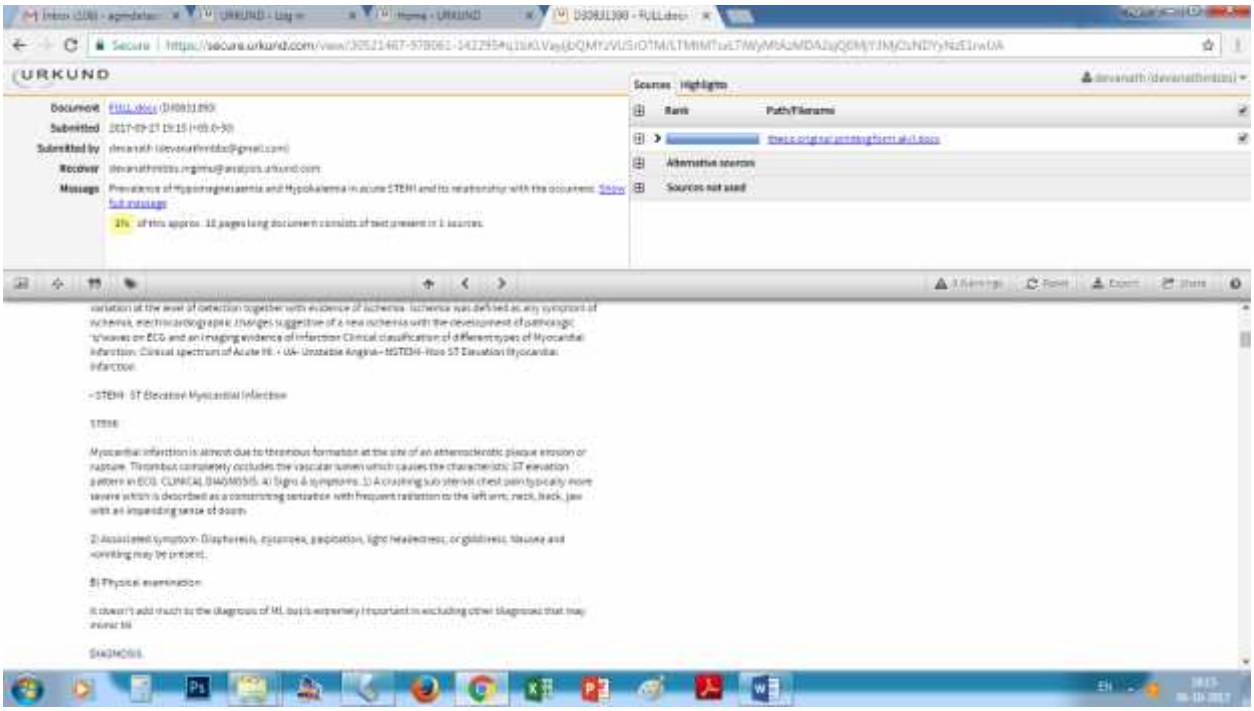
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INTRODUCTION

Ischemic heart disease causes more deaths and disability and incurs greater economic costs than any other illness in the developed world. It is the most common serious, chronic life - threatening debilitating disease in the world. Genetic factors, a high fat and energy rich diet, smoking and a sedentary life style are associated with the emergence of IHD.

With urbanization in countries with emerging economies and growing middle class, elements of the energy rich western diet are being adapted. As a result, the prevalence of risk factors for IHD and the prevalence of IHD itself are increasing rapidly. Population subgroups that appear to be particularly affected are men in South Asian countries especially India and the Middle East.

Obesity, insulin resistance and type 2 diabetes are increasing and are powerful risk factors of IHD. There is a widespread research not only for the understanding of known risk factors but also to identify new risk factors and prognosis indicators. Those are C- reactive proteins, levels, blood uric acid levels, blood magnesium levels and potassium levels. Serum magnesium and potassium levels are in the spotlight, since it leads to life threatening arrhythmias

This thesis titled "**Prevalence of Hypomagnesaemia and Hypokalemia in acute STEMI and its relationship with the occurrence of arrhythmias**" is an attempt to estimate the serum levels of magnesium and potassium in patients admitted in our ICCU department, Tirunelveli Government Medical College with AMI. Previous studies did have showed low magnesium levels in patients with acute MI and are a poor prognostic factor since it is associated with threatening arrhythmias. The prophylactic magnesium supplementation is still a controversial topic. This study is an attempt to establish the relationship between the occurrence of arrhythmias and hypokalemia and hypomagnesaemia and whether magnesium supplementation is warranted.

This study starts with the aims and objective of the study, followed by a review of literature regarding ischemic heart disease, magnesium and heart, various types of arrhythmias. Then followed by discussion of the methods and materials used and discussion regarding the observation and results followed by conclusion and summary of the study.

AIMS AND OBJECTIVES

1. To estimate the prevalence of hypomagnesaemia and hypokalemia in patients who present with AMI- STEMI.
2. To determine whether the hypomagnesaemia and hypokalemic patients are at risk for ventricular arrhythmias or other complications.

REVIEW OF LITERARURE

Ischemic heart disease is a condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium. It typically occurs when there is an imbalance between myocardial oxygen supply and demand. The most common cause of myocardial ischemia is atherosclerotic disease of an epicardial coronary artery which causes reduction in blood flow and inadequate

tissue perfusion.

Patient with ischemic heart disease fall into two large groups:

1. Coronary artery disease, which presents as stable angina.
2. Acute myocardial Infarction.

Acute myocardial Infarction:

Definition:

A 2007 expert consensus document redefined Acute MI as the detection of a rise or and fall in cardiac troponin with at least one value above the 99th URL utilizing an array with <10% coefficient of variation at the level of detection together with evidence of ischemia. [1]

Ischemia was defined as any symptom of ischemia, electrocardiographic changes suggestive of a new ischemia with the development of pathologic ‘q’waves on ECG and an imaging evidence of infarction [2]

Clinical classification of different types of Myocardial Infarction:

Clinical spectrum of Acute MI:

- UA- Unstable Angina
- NSTEMI- Non ST Elevation Myocardial Infarction
- STEMI- ST Elevation Myocardial Infarction

STEMI:

Myocardial infarction is almost due to thrombus formation at the site of an atherosclerotic plaque erosion or rupture. Thrombus completely occludes the vascular lumen which causes the characteristic ST elevation pattern in ECG.

CLINICAL DIAGNOSIS:

A) Signs & symptoms:

1) A crushing sub sternal chest pain typically more severe which is described as a constricting sensation with frequent radiation to the left arm, neck, back, jaw with an impending sense of doom.

2) Associated symptom:

Diaphoresis, dyspnea, palpitation, light headedness, or giddiness. Nausea and vomiting may be present.

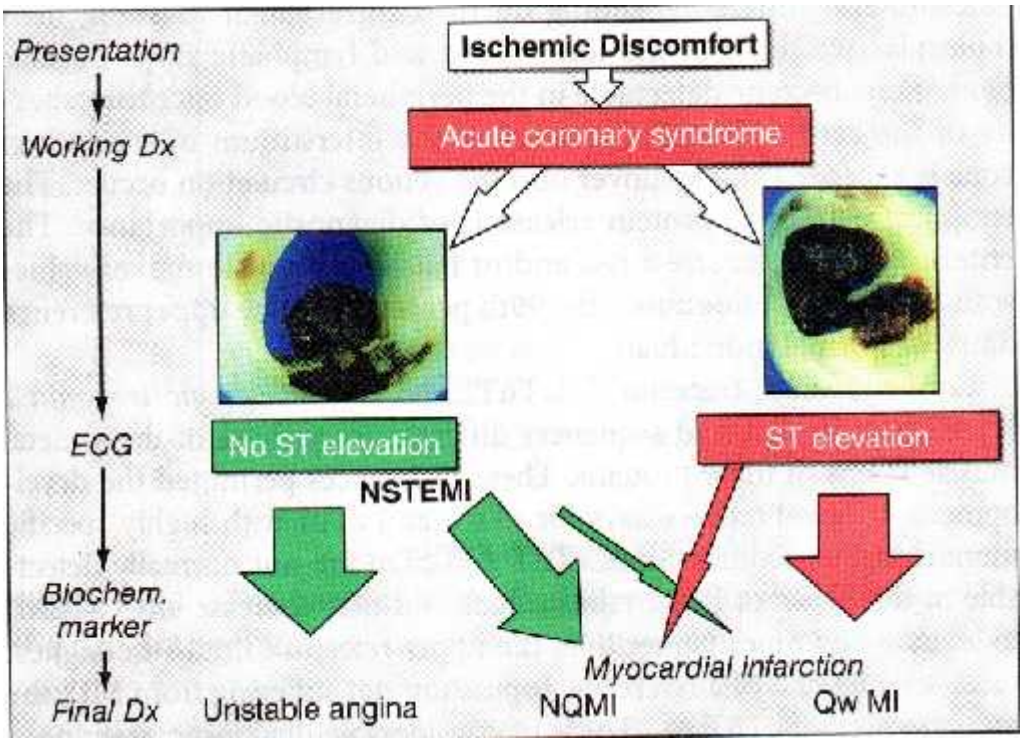
B) Physical examination:

It doesn't add much to the diagnosis of MI, but is extremely important in excluding other diagnoses that may mimic MI.

DIAGNOSIS:

ECG is a pivotal diagnostic and triage tool because it is at the centre of the decision pathway for management.

There is ST segment elevation initially in two or more contiguous leads, followed by loss of R wave amplitude in the next fours. T wave inversion occurs within a few days followed by a well-established q wave over some days to weeks.



BIOMARKERS:

Biomarkers are Creatinine kinase – cardio specific isoform (CK-MB) and Troponin–T and Troponin-I.

CPK-MB starts to rise at six hours and peaks at 12 hours and falls to normal within 3 days. Cardiac troponins are elevated after six hours and remain elevated up to two weeks.[3]

It helps in differentiating NSTEMI from UA. They are also important in assessing the progression of STEMI.

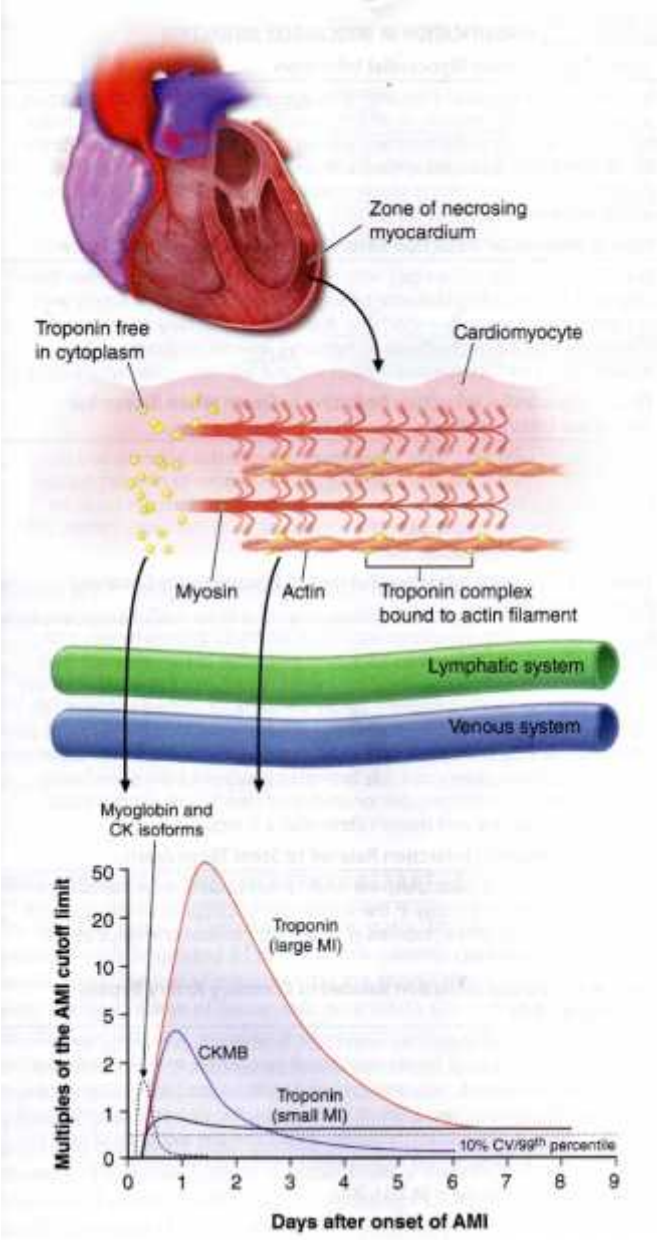


Figure showing the relationship between the myocyte injury and elevation of cardiac biomarkers like Troponin-I and T, creatinine phosphokinase and its concentration in serum in relationship to days after the onset of MI.

MANAGEMENT:

General principles:

The patient is admitted in an Intensive Coronary Care Unit. Nasal oxygen should be given. IV access should be obtained immediately.

Aspirin is essential in the management of patients with suspected STEMI and is effective across the entire spectrum of Acute Coronary Syndromes. It is given in a dose of 160-325 mg tablet in the ED, followed by 75-162mg daily oral administration along with 40-80mg Atorvastatin.

Reperfusion therapy:

It is achieved with either fibrinolytic therapy or primary percutaneous intervention or bypass grafting

Fibrinolysis:

If no contraindications are present, fibrinolytic therapy should be initiated with 30 minutes of presentation. The principal goal of fibrinolysis is prompt restoration of full coronary arterial patency.

Fibrinolytic agents:

- ✓ tPA- tissue Plasminogen Activator
- ✓ streptokinase

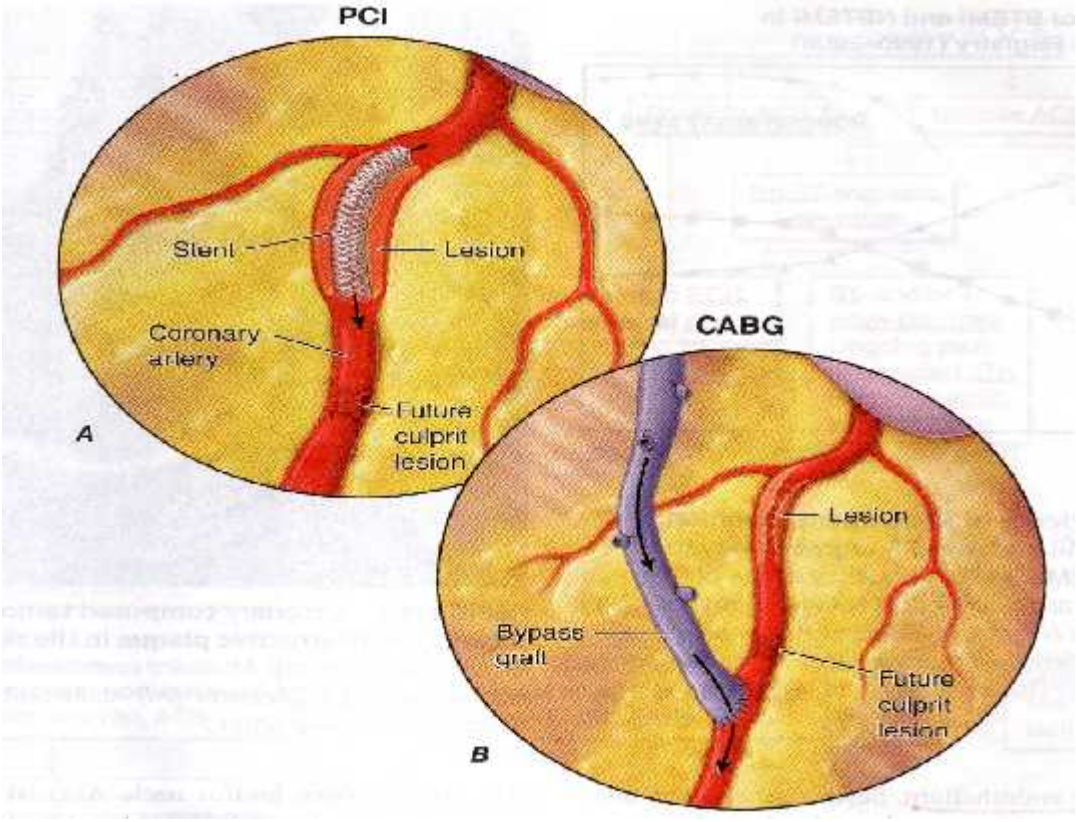
- ✓ Tenecteplase(TNK)
- ✓ Reteplase(rPA)

PRIMARY PERCUTANEOUS CORONARY INTERVENTION (PCI):

PCI, usually angioplasty and/or stenting without preceding fibrinolysis, referred to as Primary PCI is effective in restoring perfusion in STEMI.

CORONARY ARTERY BYPASS GRAFTING:

It is directed at the epicardial vessel, including the culprit lesion or lesions and future culprits, proximal to the insertion of a vein graft, a difference that may account for the superiority of CABG.



ADJUNCTIVE THERAPIES:

Nitrates:

In relieving pain and the treatment of Acute left ventricular Failure.

Beta blockers:

In reducing pain and in preventing arrhythmias during the first 12 hours.

ACE inhibitors:

It prevents Left Ventricular remodeling.

Morphine:

2 to 4 mg IV can be used for refractory chest pain that is not responsive to nitroglycerin. Adequate analgesia decreases levels of circulating catecholamines and reduces myocardial oxygen consumption.[4]

COMPLICATIONS FOLLOWING MI:

- Recurrent chest pain
- Acute pericarditis
- Dressler's syndrome
- Arrhythmias
- Cardiogenic shock

- ❖ Heparin Induced Thrombocytopenia (HIT).

Mechanical complications:

- LV aneurysm
- Ventricular Pseudo aneurysm
- Free wall rupture
- Papillary muscle rupture
- Ventricular septal rupture
- Ischemic MR (MR)

ARRYTHMIAS:

The incidence of arrhythmias after STEMI is higher in patients seen early after the onset of symptoms.

Since most deaths from arrhythmias occur during the first few hours after infarction, the effectiveness of treatment directly relates to the speed with which patients come under medical observation.

The mechanisms responsible for infarction related arrhythmias include autonomic nervous system imbalance, electrolyte disturbances, ischemia and slowed conduction in zones of ischemic myocardium.

EXACERBATING CONDITIONS:

- Electrolyte disturbances. (Hypokalemia and hypomagnesaemia)
- Hypoxia
- Acidosis
- Adverse drug effects. (Digoxin and Quinidine)

TYPES OF ARRHYTHMIAS:

1) **INTRAVENTRICULAR CONDUCTION DELAY:**

The left anterior fascicle is most commonly affected because of isolated coronary blood supply.

Bi-Fascicular and Tri-fascicular block may progress to complete heart block and other rhythm disturbances.

Treatment: None.

2) SINUS BRADYCARDIA:

Sinus Bradycardia is common in patients with RCA infarcts.

In the absence of hypotension or significant ventricular ectopic, observation is indicated.

Treatment: None

Atropine 0.5/temporary pacing mg

3) AV BLOCK:

Usually temporary. If there is hypotension or second/ third degree block, a temporary pacemaker should be considered.

4) SINUS TACHYCARDIA:

Sinus tachycardia is common in patients with acute MI and is often due to enhanced sympathetic activity resulting from pain, anxiety, hypervolemia, heart failure or fever.

Persistent ST suggests poor underlying ventricular function and is associated with excess mortality.

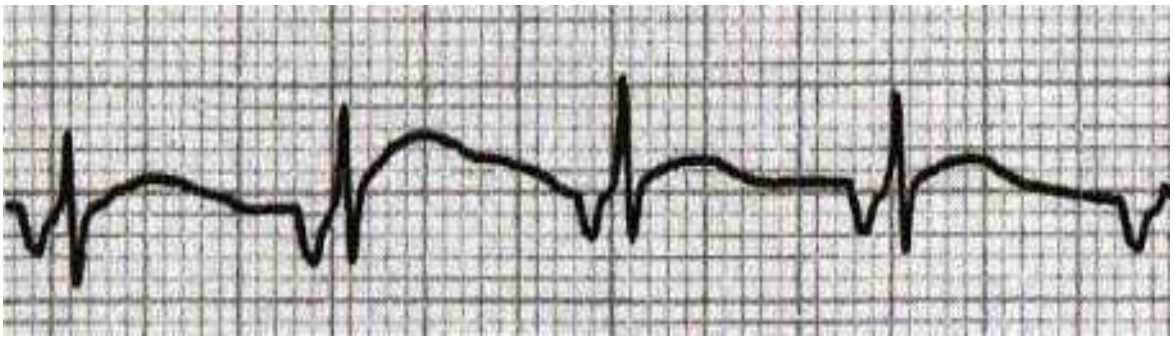
Treatment: None

5) ATRIAL FIBRILLATION:

Atrial fibrillation and flutter are observed in up to 20% of patients with Acute MI. It is usually transient. If severe enough to cause hypotension, DC cardio-conversion is required.

6) ACCELERATED JUNCTIONAL RHYTHM:

It occurs in conjunction in inferior wall MI and also seen in Digoxin intoxication.



7) VPC:

It is the common in the course of acute MI. Prophylactic treatment with lidocaine or other anti-arrhythmic has been associated with increased overall mortality and is not recommended.

8) AIVR:

It is commonly seen within 48 hours of successful reperfusion and is not associated with an increased incidence of adverse outcomes.

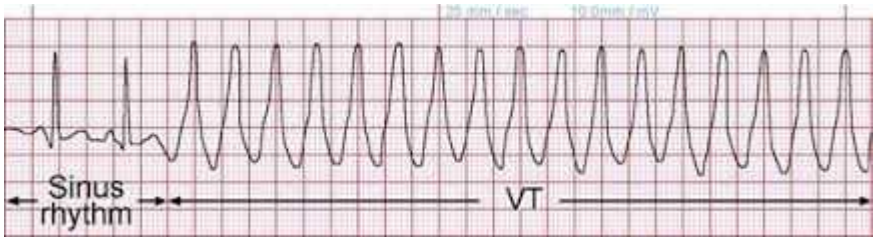
If hemodynamically unstable, sinus activity may be restored with atropine or temporary atrial pacing.

9) VENTRICULAR TACHYCARDIA (VT):

Non Sustained Ventricular Tachycardia (NSVT <30 seconds) is common in the first 24 hours after MI and is only associated with increased mortality when occurring late in the post MI course.

Sustained VT >30 seconds, during the first 48 hours, after acute MI and is associated with increased in hospital mortality.

Treatment: Cardio-version for sustained VT. Lidocaine or amiodarone for 24-48 hours.

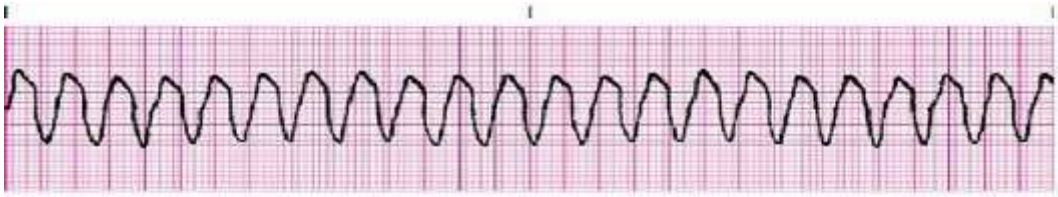


10) VENTRICULAR FIBRILLATION (VF):

VF occurs in 5% of patients with acute MI in the early hours and is life threatening.

Treatment: Unsynchronized Cardio-version.

Lidocaine or amiodarone for 24-48 hours.



MAGNESIUM:

Magnesium is the most prevalent intracellular divalent cation and the second most prevalent cation in the body [5]. The normal adult body content is approximately 20-25 g and its distribution is between 60 to 70% in bones, 25 to 30% in muscles, 6 to 8% in soft tissues and 1% in the extra cellular fluid. In

children about one-third of Mg resides on the surface of bone probably serving as a reservoir to maintain the extracellular concentration but in adults this mostly an integral part of bone crystal structure [6]. In the plasma, 55% of Mg is ionized or free, 15% is complexed to anions and the rest is bound to protein, chiefly albumin. Mg is contained within all intracellular compartments. It is principally bound to ATP (80 to 90%) and other negatively charged molecules. Total cellular Mg ranges from 5-20 mM depending on the metabolic activity of a cell. Mg is actively transported into and out of cells and is influenced by various hormonal and pharmacological factors which perhaps regulate the intracellular Mg^{2+} concentration[7]

FUNCTIONS:

The physiological role of Mg is principally related to enzyme activity; over 300 enzyme systems particularly Kinases are dependent on the presence of this Cation. This includes all enzyme utilizing ATP, they requires Mg for substrate formation [8]. Intracellular free Mg^{2+} also acts as an allosteric activator of enzyme action including critical enzyme systems such as adenylate cyclase, phospholipase C and Na/K-ATPase. It is established that Mg is critical for a number of cellular functions including oxidative phosphorylation, glycolysis, DNA transcription and protein synthesis and the clinical complications of Mg depletion are may be due to perturbation of Mg-

requiring enzyme systems. Magnesium is fundamentally required for the energy transfer reactions involving high energy compounds like ATP and creatine phosphate and thus muscle contraction. Thus, it plays vital role in heart and skeletal system function.

Transport of potassium and calcium across the plasma membrane may also require the presence of Mg. Mg has been also termed as nature's physiologic calcium channel blocker. During Mg depletion, intracellular potassium decreases while calcium and sodium increase. In view of close association of occurrence and functions of Ca and Mg, there is evidence of mutually synergistic as well as contraindicative roles of these two divalent anions, particularly in bone health and hypertension.

MAGNESIUM METABOLISM:

Magnesium is widely distributed in foods. As it is the metal ion in chlorophyll, plant products that form major source of Magnesium. Legumes and cereals are good source of Magnesium. Animal products also contain sufficient quantity [9]. Efficient mechanisms in both the gastrointestinal tract and the kidney closely regulate Mg homeostasis. Though it is absorbed along the entire intestinal tract, it appears to be most efficiently absorbed in the distal small bowel. In the intestine, an active Mg-transport system accounts

for greater fractional absorption at low dietary intake while at high dietary intakes Mg absorption occurs at a lower fractional absorption rate and is due to a passive absorption[10]. At a normal dietary Mg intake of approximately 300 to 350 mg/day, fractional absorption is 30 to 50%. This variation may be due to the presence of other nutrients interacting with Mg in the gut including high dietary fiber, phytate, oxalate, phosphate and dietary protein diets of <30 g/day which reduce Mg absorption by binding the cation or hindering absorption.

The kidney most closely regulates Mg metabolism. There exists a threshold of filtered Mg which is close to the normal plasma Mg concentration. Excessive Mg, either dietary or parenterally administered, is almost totally excreted. In contrast, at the time of Mg deprivation, the kidney avidly conserves Mg. Diet also affects renal Mg excretion, high sodium, calcium and protein diets, caffeine as well as alcohol may increase renal Mg excretion. The major site of Mg re-absorption is the thick ascending limb of Henle, which handles about 65% of the filtered load. Re-absorption in the proximal tubule, 20-30% of filtered load, is linked to sodium and water as well as calcium transport. The mechanisms of Mg transport in the intestine and kidney is still unclear.

Despite early proposals for the existence of a specific hormonal control of Magnesium homeostasis, no single endocrine factor that controls circulating or urinary Magnesium has been identified. It has been described as the body's 'orphan ion', because of an apparent lack of a specific endocrine control similar to that existing for calcium, sodium and potassium. A number of hormones, including parathyroid hormone and calcitonin, vitamin D, insulin, glucagon, antidiuretic hormone, aldosterone and sex steroids have been reported to influence Magnesium balance, notwithstanding the possibility that these may not be primary regulators of magnesium homeostasis. Recent observations suggest that these hormones act through a common second messenger, adenosine 3', 5'- cyclic mono-phosphate to enhance magnesium transport and modulate magnesium excretion at that nephron site.

MAGNESIUM AND HEART:

The rate of total cardiovascular and sudden cardiac death is associated inversely with Mg^{2+} content in drinking water i.e. water hardness.[11]

Also there is an association between death from Acute MI and a reduced Mg^{2+} concentration, the mechanisms implicated are focal myocardial necrosis, small vessel changes and spatially heterogeneous prolongation of

ventricular repolarization with induction of polymorphic VT occur in association with Mg^{2+} deficiency in animal studies.

ACUTE MYOCARDIAL INFARCTION:

A transient decline in the serum Mg^{2+} concentration takes place in 6 to 46% patients with AMI and the concentration is normalized in two weeks. The decline in the serum Mg^{2+}concentration following AMI is probably explained by a shift between magnesium compartments in the body. In this, catecholamine over secretion plays a major role [12]. Catecholamine induced lipolysis with aggregation of Mg^{2+} wit free-fatty acids leads to sequestration of Mg^{2+} in adipocytes and explains partly the observed hypomagnesaemia [13]. The catecholamine effect is not unique to AMI but all acutely stressful conditions may be accompanied by hypomagnesaemia.[14,15,16]

It has also been proposed that hypomagnesaemia and Mg^{2+} depletion ante cede the development of myocardial infarction. In support of this hypothesis, depressed myocardial Mg^{2+} concentrations have been reported in patients who have died of MI.

HEART FAILURE:

Hypomagnesaemia and Mg^{2+} depletion are frequently encountered in patients with heart failure and the presence of low Mg^{2+} indicates a worse prognosis than among patients with a normal Mg^{2+} status. Those patients with heart failure had significantly reduced mononuclear cell Mg^{2+} levels and this was independent of diuretic therapy. The patients with a low left ventricular EF and ventricular arrhythmias had decreased tissue Mg^{2+} levels together with increased QT interval depression.

Factors contributing to the genesis of hypomagnesaemia and Mg^{2+} depletion consist of:

neurohormonal activation and pharmacological treatment of the disease

(thiazide and loop diuretics)

Low cardiac output

Diminished renal flow

Activation of compensatory vaso-constrictive and volume expanding mechanisms

1) Catecholamine excretion can cause hypomagnesaemia.

2) Activation of RAAS can increase the renal excretion of Mg^{2+} indirectly.
3) Aldosterone and vasopressin induced fluid retention and consequent expansion of the extracellular volume results in reduced Mg^{2+} absorption in PCT. Volume expansion also leads to intestinal edema with reduced absorption of Mg^{2+}.[17]

As Mg^{2+} is mandatory co-factor of Na^{2+}-K^{+} ATPase, the hyperaldosteronism induced cellular Na^{2+} accumulation and K^{+} loss are insufficiently counter acted by this pump. Na^{2+}/Ca^{2+} counter transport will be increased leading to intracellular accretion of calcium. Calcium overload in the failing heart may cause additional deterioration of ventricular pump and trigger ventricular arrhythmias.[18]

CORONARY ARTERY DISEASE:

Magnesium depletion might leads to atherosclerosis. Depressed myocardial Mg^{2+} levels have been found post-mortem in patients with ischemic heart disease [19]. A low dietary magnesium intake correlates significantly with the incidence of ischemic heart disease. Mg^{2+} deficiency produces endothelial changes and constricts the coronary arteries. Intracellular Mg^{2+} deficiency is also seen in patients with coronary artery disease.

Evidence suggests that endothelial dysfunction may be the initiating event in the atherosclerotic process that subsequently leads to clinical CAD [20]. Accordingly, there has been an on-going, aggressive search for therapeutic choices suitable for reversal of endothelial dysfunction with the hope that such intervention, if instituted early in the course of the disease, might prevent or modify the subsequent risk of clinical disease and related cardiac events. Magnesium, which is an inexpensive, natural, and relatively safe element, has been shown in the present study to improve endothelial function and thus may be justified as an adjuvant therapy for CAD patients. Further studies, with larger populations, are needed to prove our findings

CARDIAC SURGERY:

Cardiac surgery where cardiopulmonary bypass is utilized leads to postoperative decline in serum Mg^{2+} levels [21]. The proposed causes for the decline in serum Mg^{2+} concentration following cardiac surgery are hemo dilution, use of diuretics, secondary hypoaldosteronism, increased anabolic activity and enhanced sympathetic activity.

ELECHOPYSIOLOGICAL EFFECTS OF MAGNESIUM

The functions of magnesium and potassium are closely related. Hypokalemia is almost always associated with hypomagnesaemia. Hypomagnesaemia is present in 40% of patients with hypokalemia [22]. Hypokalemia can be corrected only if magnesium is replete [23].

Intracellular magnesium is a potent blocker of cardiac muscle cell potassium channels. Potassium has a tendency to flow into the cell from the ECF compartment. When an electrochemical gradient favours outward flow of potassium, Mg2+ ions blocks this channel preventing the outward flow of K+. Hence these channels acquire a property known as inward rectification. Inward rectification is important in the maintenance of plateau phase of cardiac muscle action potential [24].

Magnesium ions modulate the intracellular calcium levels in the sarcoplasmic reticulum through L type calcium channels. When there is an increase or decrease of magnesium ion levels, there is a corresponding inhibition or enhancement of the calcium inflow. Thus Mg regulates the calcium channel function and transmembrane calcium flow. Magnesium is considered as nature’s physiological calcium blocker.

The mechanisms by which magnesium enters the cell and how it gets removed from the cell through a Na+ - Mg2+ exchange trans membrane protein are not clearly identified.

Arrhythmia mechanisms suppressible by magnesium

Mechanisms of arrhythmias:

1. Re-entry enhanced
2. Automaticity
3. Triggered activity

After depolarization's or EAD's and delayed after depolarization's (DAD) play an important role in triggered activity. Afterdepolarization occur during the course of repolarization. If it occurs during the phase 4 of action potential, they are called as DAD. Early after depolarization's can be stopped by magnesium.

In vitro experimental studies suggest that tachycardia produced by these mechanisms is terminated by addition of magnesium Though mechanisms behind EAD and DAD are different, this is true.

EAD is caused by enhanced Ca2+ inflow through L type Ca2+ channels. Contributory effects are made by Na+ ions flowing through non-inactivating Na2+ channels. The emergence of EAD is helped by the prolonged duration of action potential. EAD's most commonly seen in the mammalian heart purkinjie cells which have the longest duration of AP. Bradycardia or any sinus pause, substances like quinidine, barium, aconite, Bay K 8644 lengthen the duration of action potential.

Magnesium inhibits the inflow of Ca2+ ions. By this mechanism, it suppresses the EAD that occur during the phase 2 or plateau phase of the AP. It also influences the EAD's that occur during the phase 3 of membrane potential which are more negative than - 60 millivolts and are modulated by sodium currents.

The classical example of an arrhythmia based on EAD's is pause dependent "torsade de pointes" ventricular tachycardia. It is associated with a long QT interval which may be drug or toxic induced.

Delayed after depolarization's can be thought of as a result of oscillations of the resting membrane potential (RMP) of myocytes. These cardiac myocytes may be overloaded with calcium ions or they have decreased conductance of K+ ions. This leads to activation of non-selective,

inward transient flow of cations. It also activates the Na+-Ca2+ exchanger. DAD's also induced by catecholamines, digitalis toxicity, caffeine, theophylline, histamine, lysophosphatidyl choline. This triggers the activity responsible for arrhythmias which is suppressed by magnesium [25].

Myocardial magnesium depletion enhances SA node automaticity. Mg2+ supplementation has a negative chronotropic effect. It also prolongs the PR interval by its action on the AV node. The proposed mechanism is it lengthens the AV nose refractory period. It does node have any actions over the His-purkinjie conducting system. It does not increase the refractory period of the atrial myocardium or the ventricular myocardium.

ECG findings in magnesium deficiency include ST segment depression, flattering of T wave and QT prolongation.

In hypomagnesaemia, there is Bradycardia as a result of negative inotropic action. There is PR interval prolongation, intraventricular conduction abnormalities. The QRS complex is widened. The T wave are shaper and higher.

Magnesium and hypokalemia:

Observations in humans and animals show that Mg depletion renders the cell unable to retain K difference between intra- and extracellular space and results in intracellular K depletion. This phenomenon occurs because the Na/K pump action depends on Mg. The insufficient action of the Na/K pump results in K depletion and intracellular Na accumulation. Furthermore the kidney cannot retain potassium.

MATERIALS AND METHODS

Type of study:

Study population:

Patients who are admitted in Intensive Coronary Unit and were diagnosed as acute ST segment elevation myocardial infarction.

Total no of cases: 100

Diagnosis and investigation of the cases:

Diagnosis:

Patients who are admitted in ICCU with symptoms suggestive of AMI were evaluated. A complete history was elicited from the patient and detailed clinical examination was done. A standard twelve lead electrocardiogram was done in all patients. It was then analyzed for the presence of acute ST elevation myocardial infarction.

ECG CRITERIA:

1. New onset ST segment elevation more than 1mm in limb leads and or more than 2 mm in precordial leads.
2. The ST segment elevation should be present in two or more leads.
3. The two or more leads should be contiguous with respect to each other.

Continuous cardiac monitoring and standard 12 lead electrocardiogram was done to identify any arrhythmias in the first 24 hours since admission.

INCLUSION CRITERIA:

1. All patients with definite evidence of acute coronary syndrome-STEMI as diagnosed by chest pain<24 hrs., ECG, enzyme assays and ECHO.
2. All patients with definite evidence of arrhythmias as diagnosed by Continuous Cardiac monitoring and standard ECG.
3. Significant arrhythmia's causing hemodynamic instability, sustained Palpitation, syncope.

EXCLUSION CRITERIA:

1. Use of loop and thiazide diuretics
2. Poor dietary intake/malnourished state.
3. Chronic diarrhea/ persistent vomiting.
4. Diabetic ketoacidosis
5 Malabsorption syndromes.

PROCEDURE DONE

1. Informed consent is obtained from the patient for the study.
2. Around 3 ml of venous blood sample is collected from the patient.
3. The time interval between admission and sample collection did not exceed 6 hours.
4. The sample was transferred to a plain tube without any anticoagulants.
5. Proper labeling was done.
6. The proforma for each patient was filled appropriately.
7. The sample was sent to the biochemistry laboratory.
8. It is separated into the serum and fibrin clot.
9. The sample was centrifuged at 2000 rpm for the separation serum from the blood.
10. The serum obtained was estimated for its magnesium concentration using

    Colorimetric method.
11. Through continuous cardiac monitoring and standard ECG at regular intervals, arrhythmias were identified.

A total of 100 patients who met the inclusion criteria were investigated for the serum magnesium and potassium levels within six hours since admission and overlooked for the presence of arrhythmias during the first 48 hours.

Method for magnesium estimation:

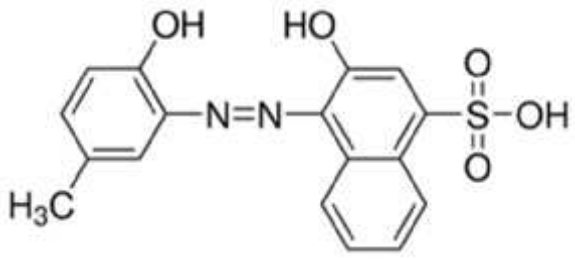
Colorimetry:

The concentration of coloured solutes in a solution is determined by a technique called colorimetry. A light of known wavelength and intensity is passed through the coloured solution. After the light passes through the solution there is a change in the wavelength and may be the intensity of the light beam. These properties are detected by spectrophotometer. The change in the property of the light emitted and detected is proportional to the intensity of the colour in the solution. The intensity of the colour is directly proportional to the concentration of the solute. Thus, indirectly we can estimate the concentration of the dissolved compound.

Calmagite method:

It is a complex chemical compound. It becomes wine red in colour when it combines with a metal ion like Mg2+ and blue in colour when it is not combined with a metal ion.

Structure:



Principle:

When magnesium combines with calmagite in the presence of an alkaline medium it imparts a red colour. Calcium and protein interference is eliminated by the addition of specific chelating agents and detergents. The intensity of the colour formed is directly proportional to the concentration of magnesium present in the sample.

Magnesium + calmagite → red coloured complex

(alkaline medium)

Normal reference values:

Serum:

Children: 1.5 to 2 mg/dl

Adults: 1.7 to 2.5 mg/dl

CSF: 2.0 t0 3.0 meq/dl

Urine: 6.0 to 8.5 meq/dl

Sample material:

Serum free hemolysis. Magnesium is stable in plasma for 7 days at 2-8 degree Celsius.

Procedure:

Wavelength/filter: 510 nm /green

Temperature: room temperature

Light path: 1 centimeter

Incubation time: 5 minutes

Linearity:

The procedure is linear up to 10 meq/l. the sample should be diluted with distilled water if this value is exceeded. The assay should be repeated. The value is corrected using an appropriate dilution factor.

Calculations:

Magnesium in meq/l = (Abs.T / Abs.S) *2

SERUM POTASSIUM ESTIMATION:

Ion-selective electrode (ISE) methods use a glass ion-exchange membrane for sodium and a valinomycin neutral-carrier membrane for potassium measurement.

METHOD USED:

Ion- selective electrode.

SAMPLE:

Serum free from hemolysis.

Plasma and serum sodium and potassium are stable for at least 1 week at room or refrigerator temperatures and for at least 1 year frozen. Urine sodium and potassium are stable for at least 1 week at room temperature and indefinitely if frozen.

There are two general types of ISE measurements made on clinical samples. “Direct” potentiometric systems measure the ion activity in an undiluted sample. “Indirect” ISE systems measure the ion activity in a prediluted sample. Because ISE measurements determine the activity of an ion in the water-volume fraction in which it is dissolved, “direct” measurements are unaffected by conditions such as hyperproteinemia or hyperlipidemia,

which alter the volume fraction of water in serum. However, “indirect” methods are usually sensitive to this physiological effect because the dilution step itself is based on total volumes, and after dilution, the volume occupied by soluble serum molecules becomes insignificant with respect to the total diluent volume.

NORMAL SERUM LEVELS:

3.5 to 5 meq/l.

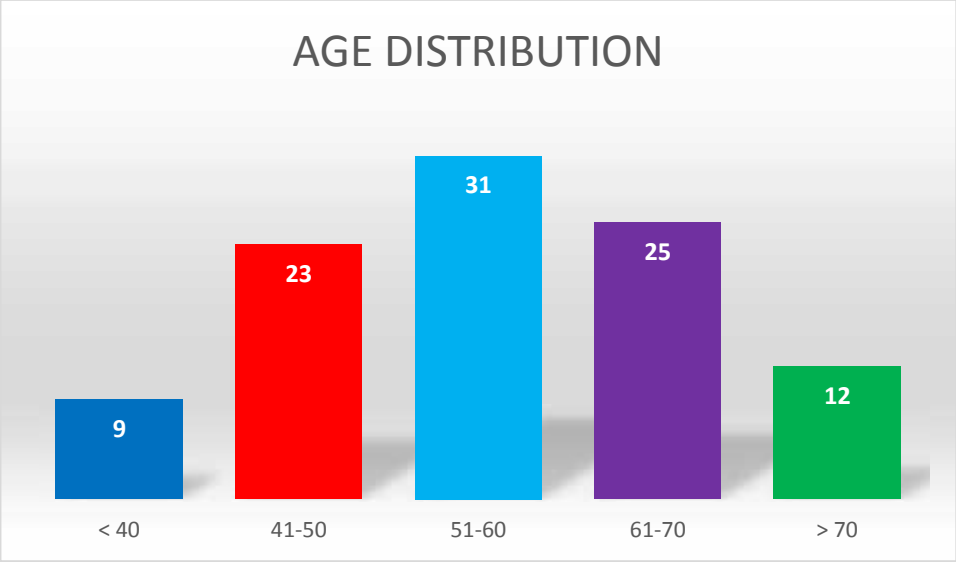
URINE:

Potassium 25 to 125 mmol/24 hr.

OBSERVATION & RESULTS

Age Distribution:

AGE(IN YEARS)	NO OF PATIENTS
< 40	9
41-50	23
51-60	31
61-70	25
> 70	12



Range- 34-77 years

Mean ± S.D = 57.65 ± 12.05

Most commonly patients were in 6th decade

SEX DISTRIBUTION:

SEX	NO OF PATIENTS
MALE	83
FEMALE	17

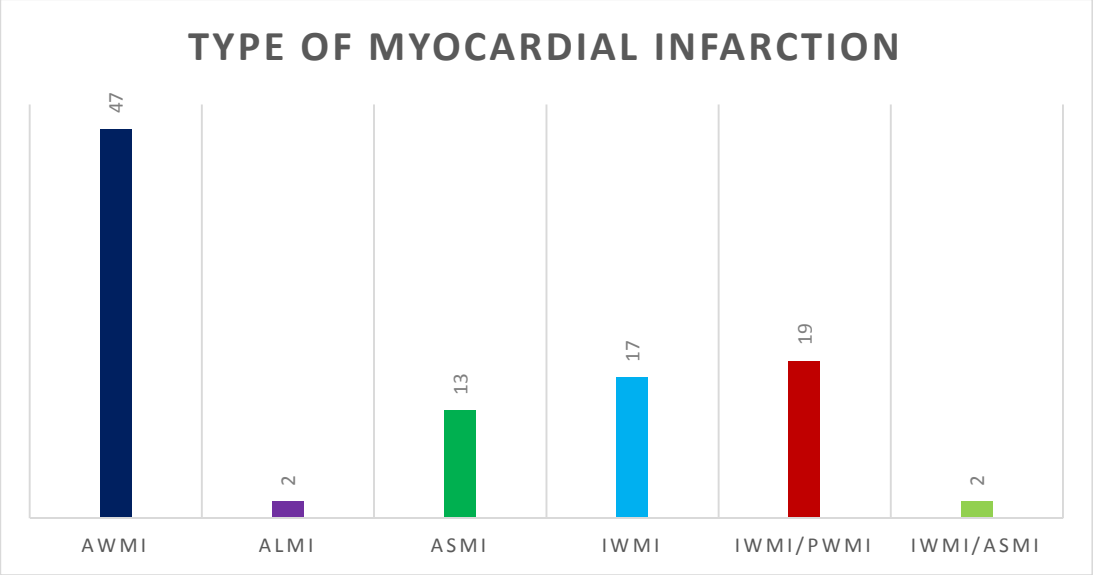
MALE: FEMALE RATIO = 5.1:1

Acute myocardial infarction is most commonly seen in males. N=83

TYPE OF MYOCARDIAL INFARCTION

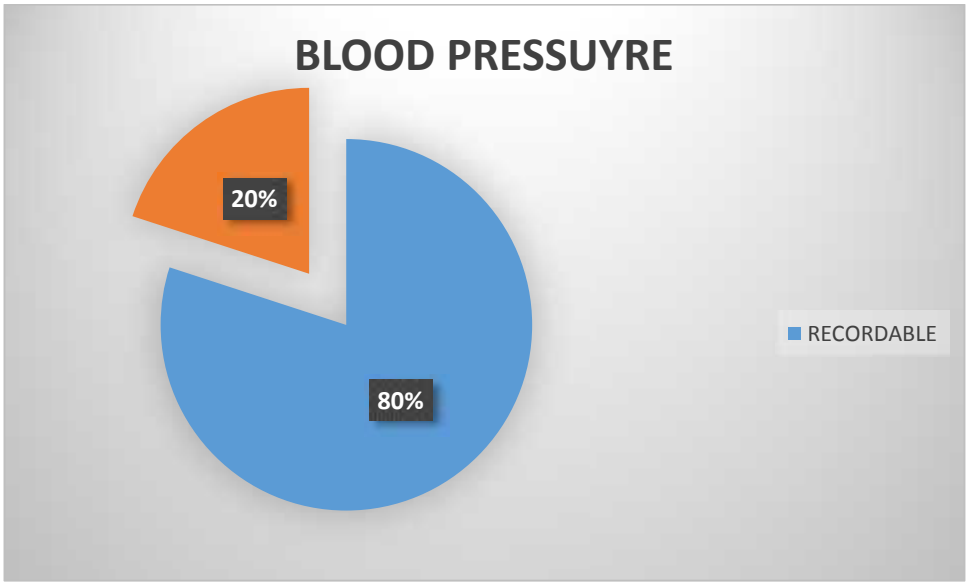
TYPE OF MI	NO OF PATIENTS
AWMI	47
ALMI	2
ASMI	13
IWMI	17
IWMI/PWMI	19
IWMI/ASMI	2

AWMI (N = 47) IS MOST COMMON TYPE FOLLOWED BY IWMI WITH PWMI



BLOOD PRESSURE

BLOOD PRESSURE	NO OF PATIENTS
RECORDABLE	80
NOT RECORDABLE	20

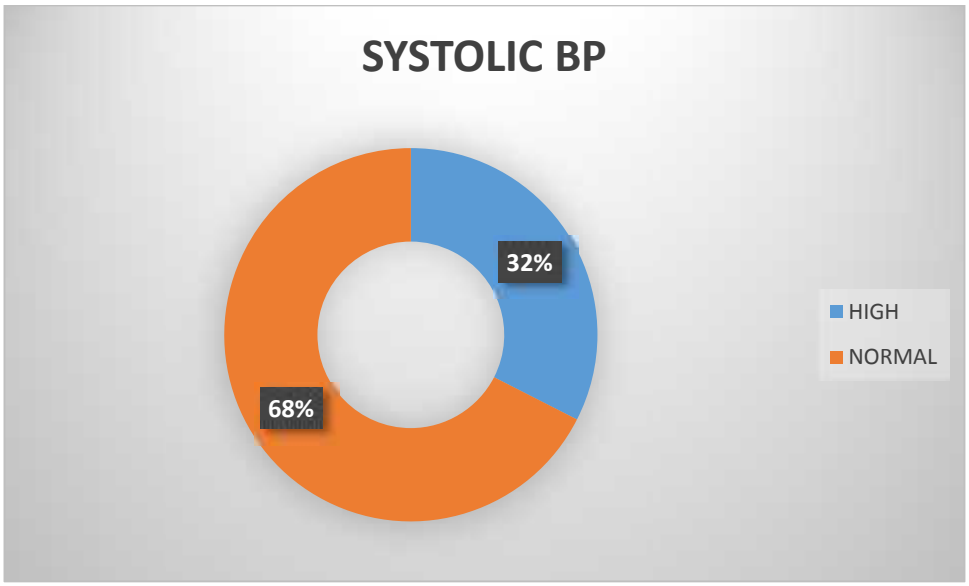


20 % of the patients with AMI associated with arrhythmias presented with hemodynamic instability.

SYSTOLIC BLOOD PRESSURE

SYSTOLIC BP(N-80)	NO OF PATIENTS	PERCENTAGE
HIGH	26	32.50%
NORMAL	54	67.50%

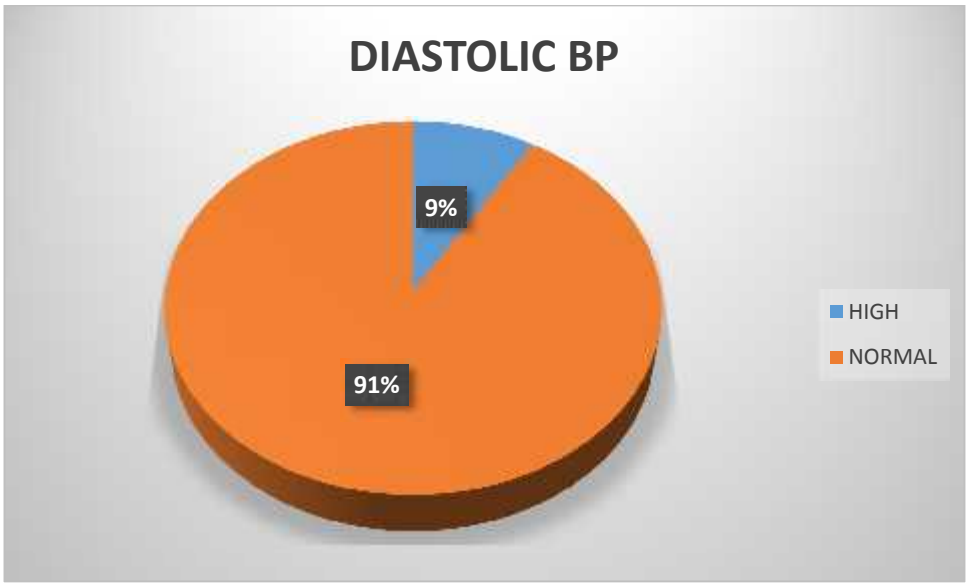
AMONG PATIENTS FOR WHOM BP IS RECORDABLE 26 PATIENTS (32.50%) HAS HIGH LEVEL



DIASTOLIC BLOOD PRESSURE

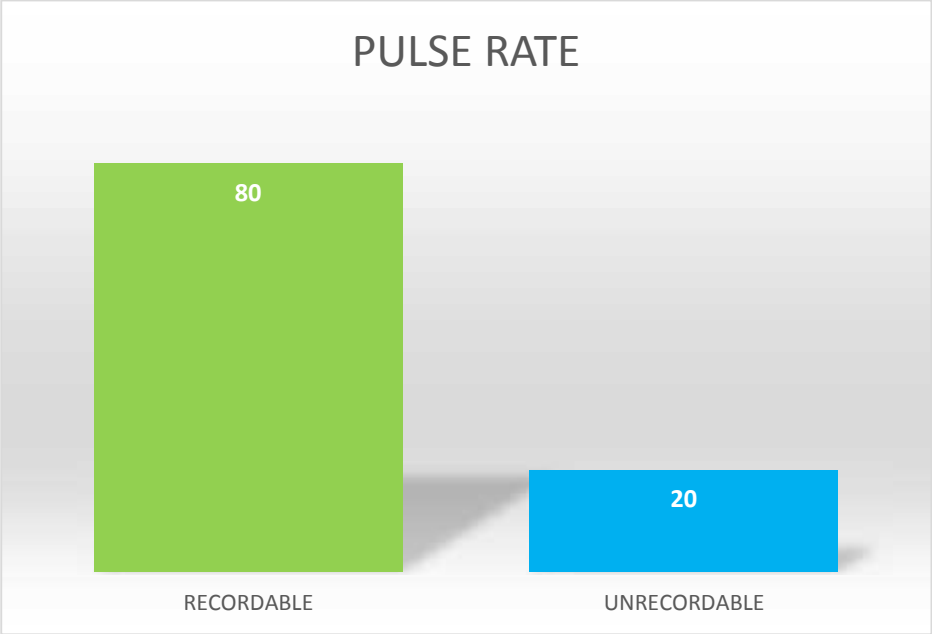
DIASTOLIC BP (N=80)	NO OF PATIENTS	PERCENTAGE
HIGH	7	8.75%
NORMAL	73	91.25%

AMONG PATIENTS FOR WHOM BP IS RECORDABLE 7 PATIENTS (9%) HAS HIGH LEVEL

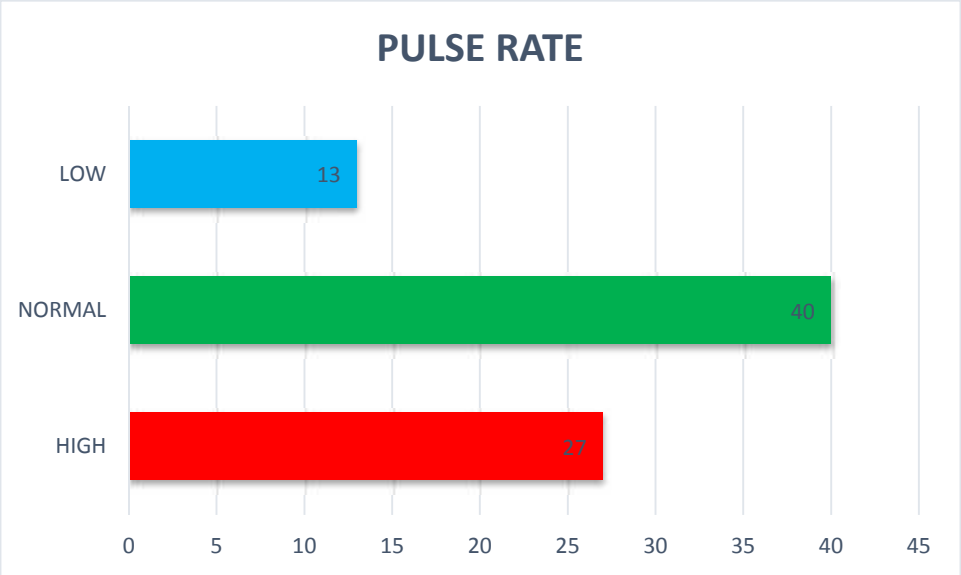


PULSE RATE

PULSE RATE	NO OF PATIENTS
RECORDABLE	80
UNRECORDABLE	20

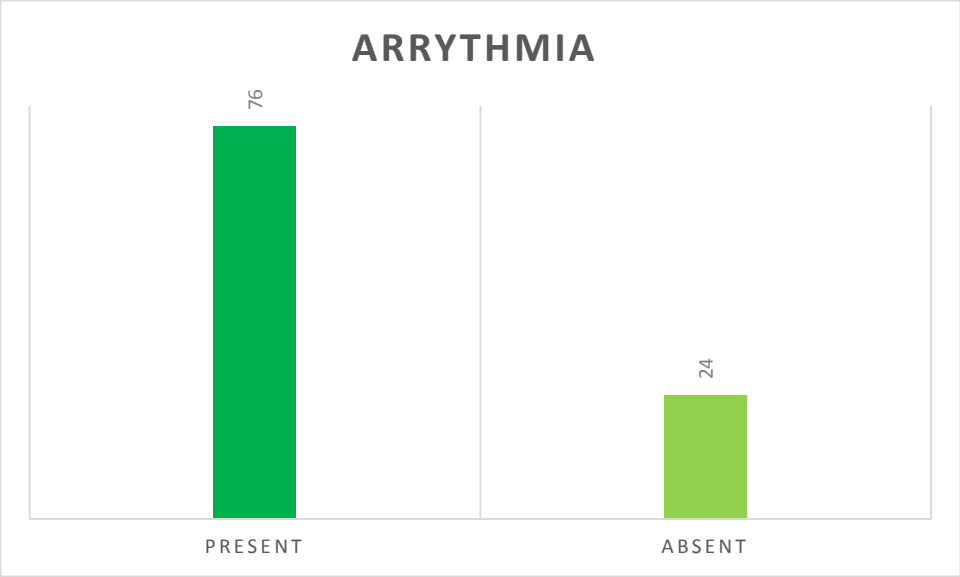


PULSE RATE(N-80)	NO OF PATIENTS	PERCENTAGE
HIGH	27	33.75%
NORMAL	40	50%
LOW	13	16.25%

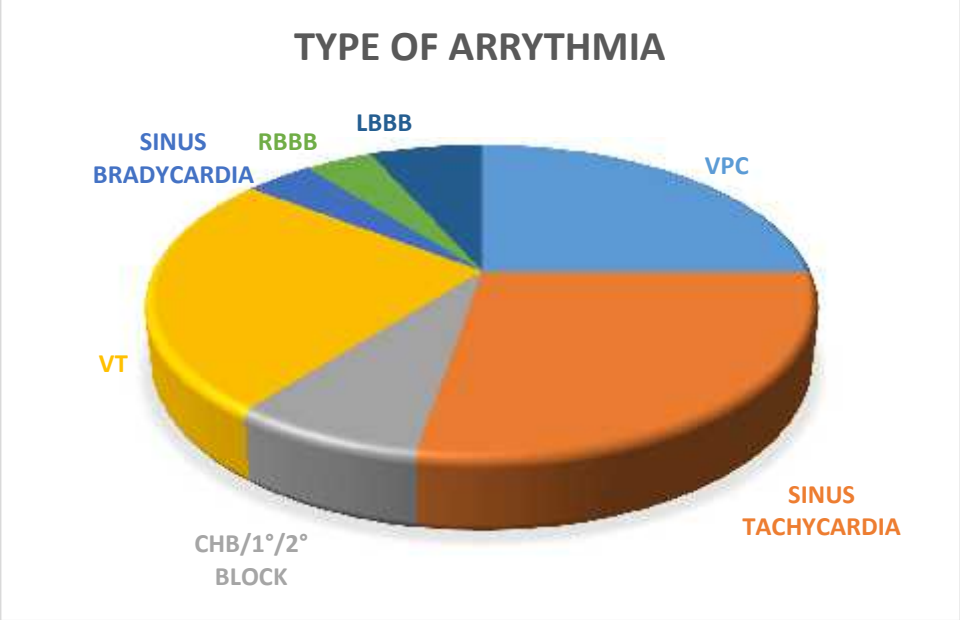


ARRYTHMIA

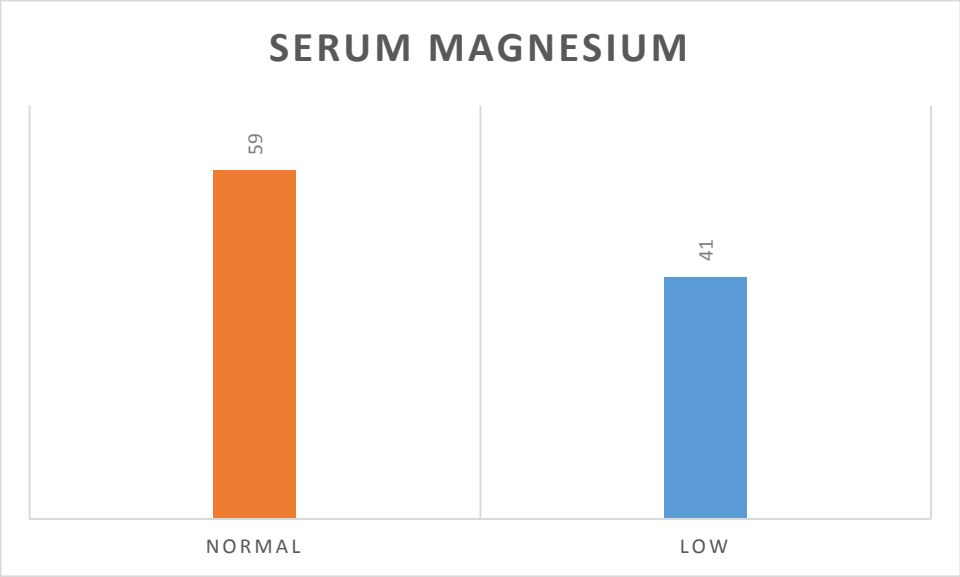
ARRYTHMIA	NO OF PATIENTS
PRESENT	76
ABSENT	24



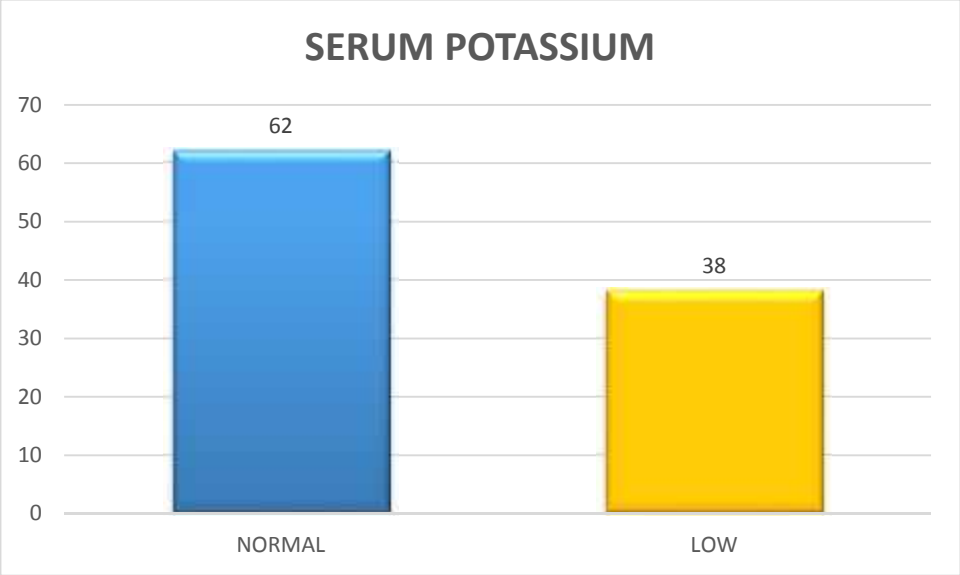
TYPE OF ARRYTHMIA	NO OF PATIENTS (N=76)	PERCENTAGE
VPC	19	25%
SINUS TACHYCARDIA	21	27.6%
CHB/1°/2° BLOCK	6	7.9 %
VT	19	25%
SINUS BRADYCARDIA	3	3.9%
RBBB	3	3.9%
LBBB	5	7.7%



SERUM MAGNESIUM	NO OF PATIENTS
NORMAL	59
LOW	41

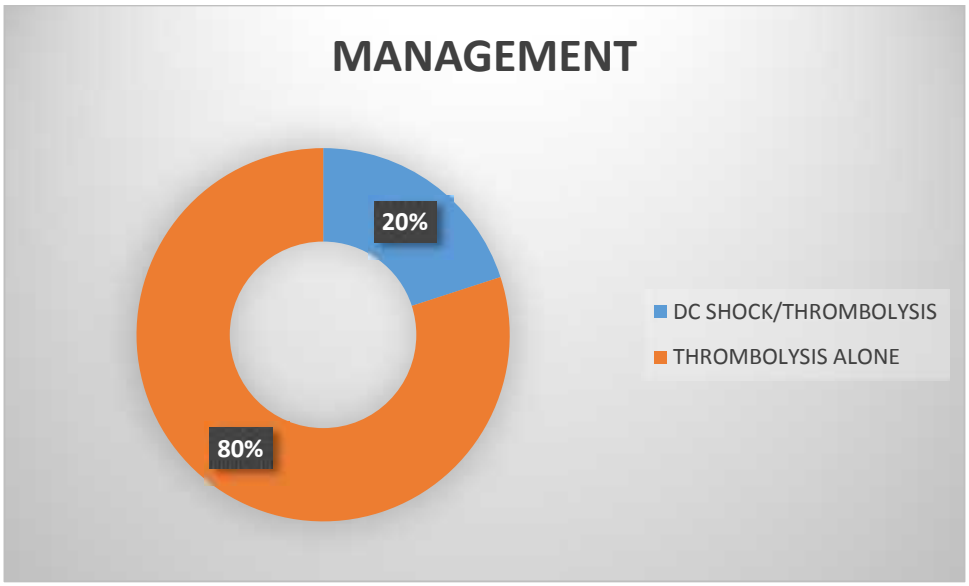


SERUM POTASSIUM	NO OF PATIENTS
NORMAL	62
LOW	38

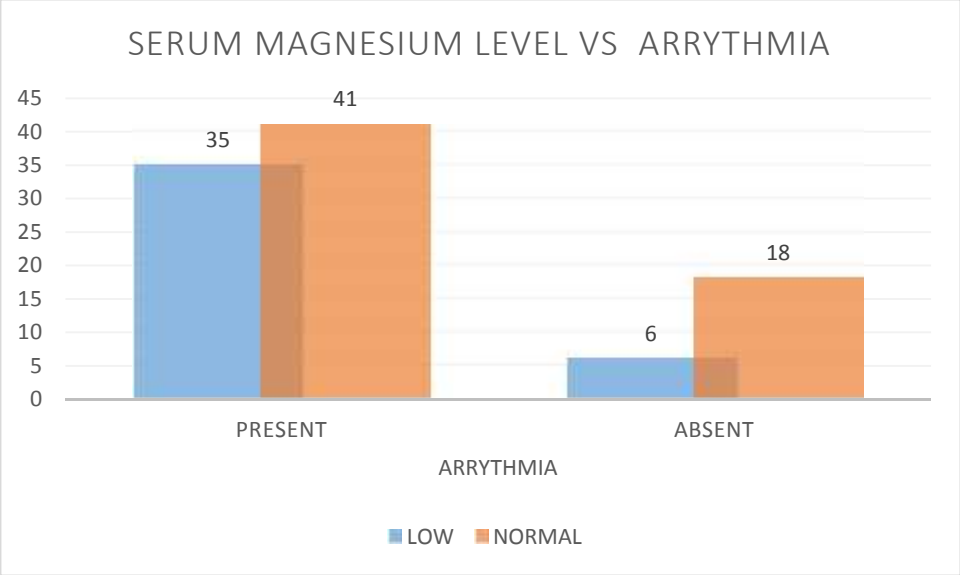


TREATMENT OF PATIENTS

MANAGEMENT	NO OF PATIENTS
DC SHOCK/THROMBOLYSIS	20
THROMBOLYSIS ALONE	80

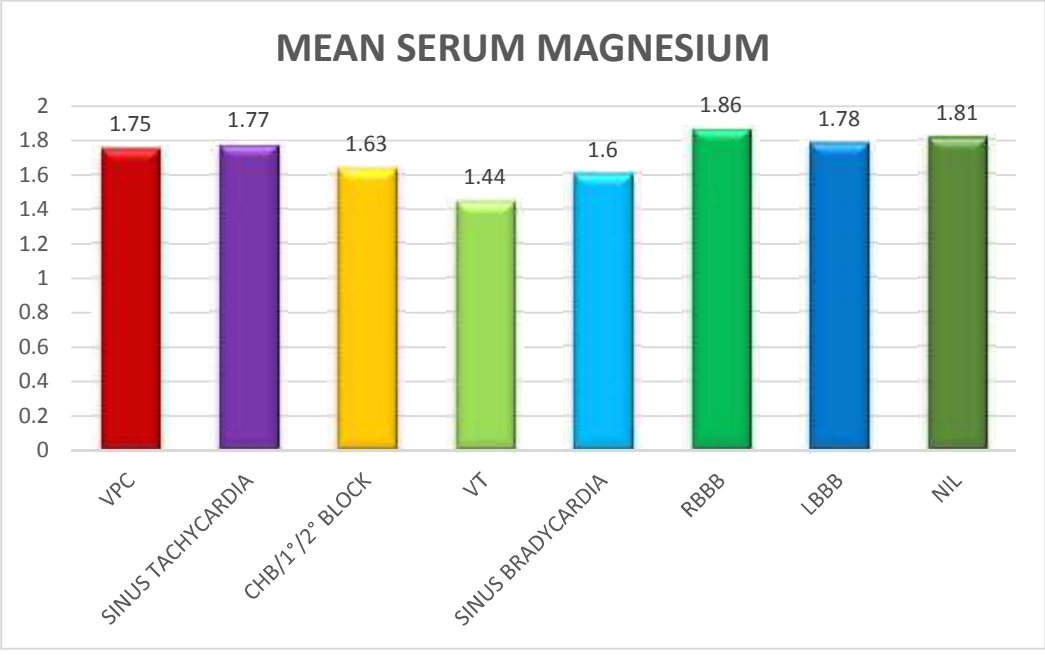


SERUM MAGNESIUM	ARRYTHMIA	
	PRESENT	**ABSENT**
LOW	35	6
NORMAL	41	18
MANN WHITNEY U TEST		
P VALUE - 0.048		
SIGNIFICANT		

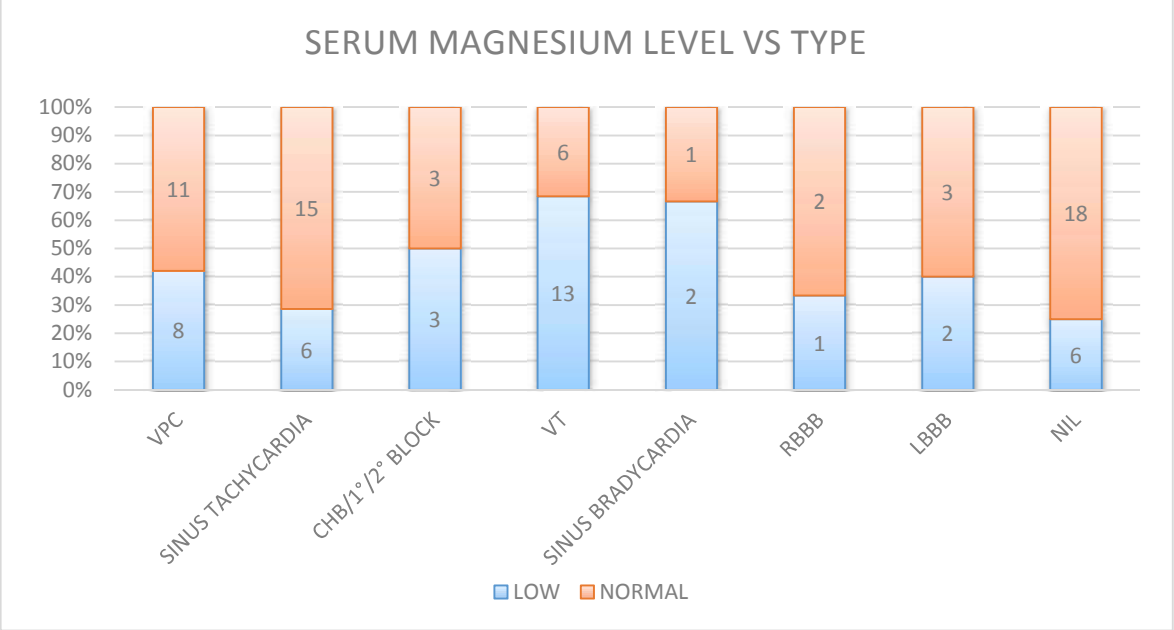


TYPE OF ARRYTHMIA	SERUM MAGNESIUM	
	MEAN	STANDARD DEVIATION
VPC	1.75	0.27
SINUS TACHYCARDIA	1.77	0.2
CHB/1°/2° BLOCK	1.63	0.2
VT	1.44	0.48
SINUS BRADYCARDIA	1.6	0.1
RBBB	1.86	0.32
LBBB	1.78	0.28
NIL	1.81	0.25

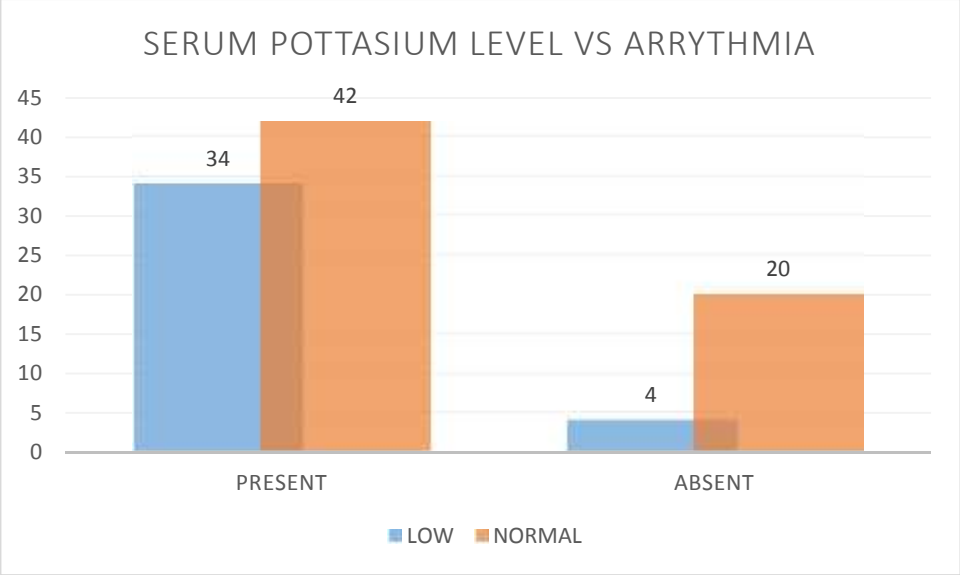
VT HAS LOW MEAN SERUM MAGNESIUM WHICH IS STATISTICALLY SIGNIFICANT WITH P VALUE OF 0.008



	SERUM MAGNESIUM	
TYPE OF ARRYTHMIA	**LOW**	**NORMAL**
VPC	8	11
SINUS TACHYCARDIA	6	15
CHB/1°/2° BLOCK	3	3
VT	13	6
SINUS BRADYCARDIA	2	1
RBBB	1	2
LBBB	2	3
NIL	6	18



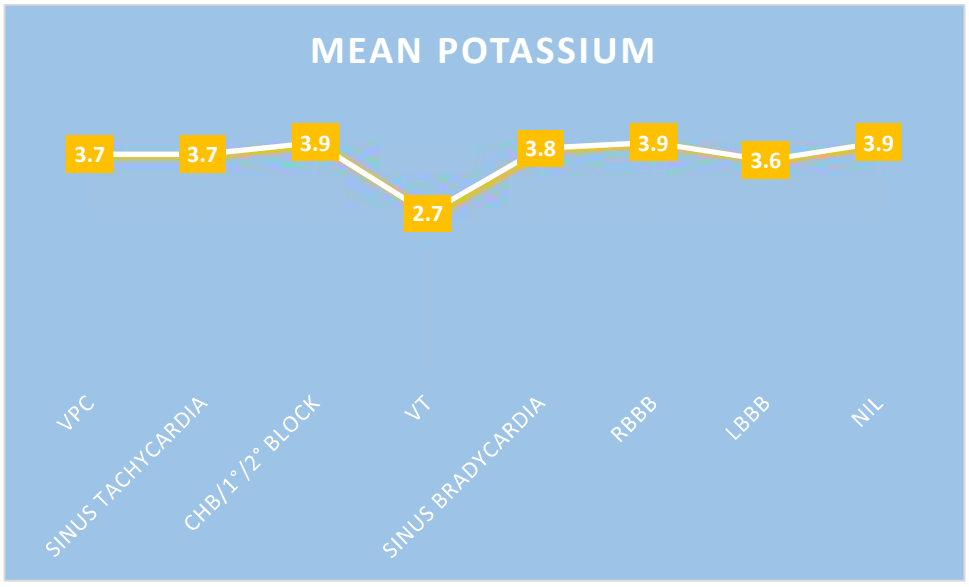
SERUM POTASSIUM	ARRYTHMIA	
	PRESENT	ABSENT
LOW	34	4
NORMAL	42	20
MANN WHITNEY U TEST		
P VALUE - 0.013		
SIGNIFICANT		



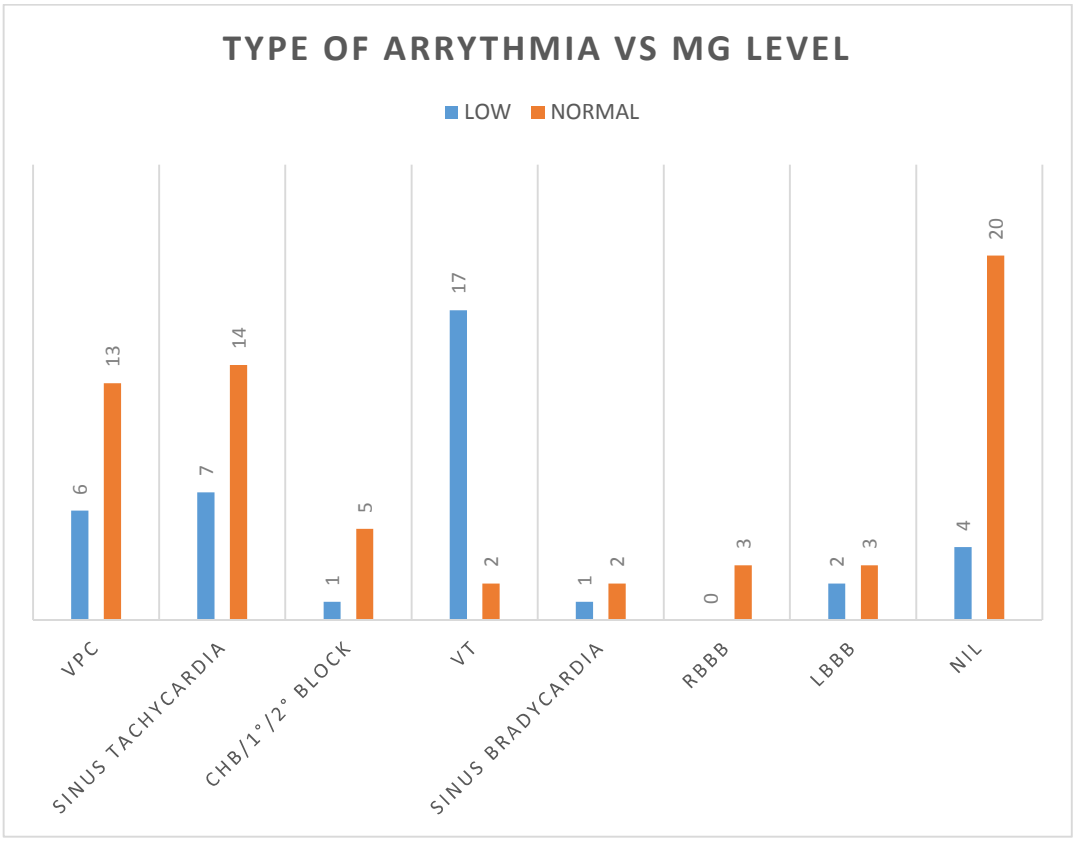
TYPE OF ARRYTHMIA	SERUM POTASSIUM	
	MEAN	STANDARD DEVIATION
VPC	3.7	0.54
SINUS TACHYCARDIA	3.7	0.4
CHB/1°/2° BLOCK	3.9	0.51
VT	2.7	0.54
SINUS BRADYCARDIA	3.8	0.52
RBBB	3.9	0.36
LBBB	3.6	0.36
NIL	3.9	0.45

VT HAS LOW MEAN SERUM POTASSIUM WHICH IS

STATISTICALLY SIGNIFICANT WITH P VALUE OF 0.001



TYPE OF ARRYTHMIA	SERUM POTASSIUM	
	LOW	NORMAL
VPC	6	13
SINUS TACHYCARDIA	7	14
CHB/1°/2° BLOCK	1	5
VT	17	2
SINUS BRADYCARDIA	1	2
RBBB	0	3
LBBB	2	3
NIL	4	20



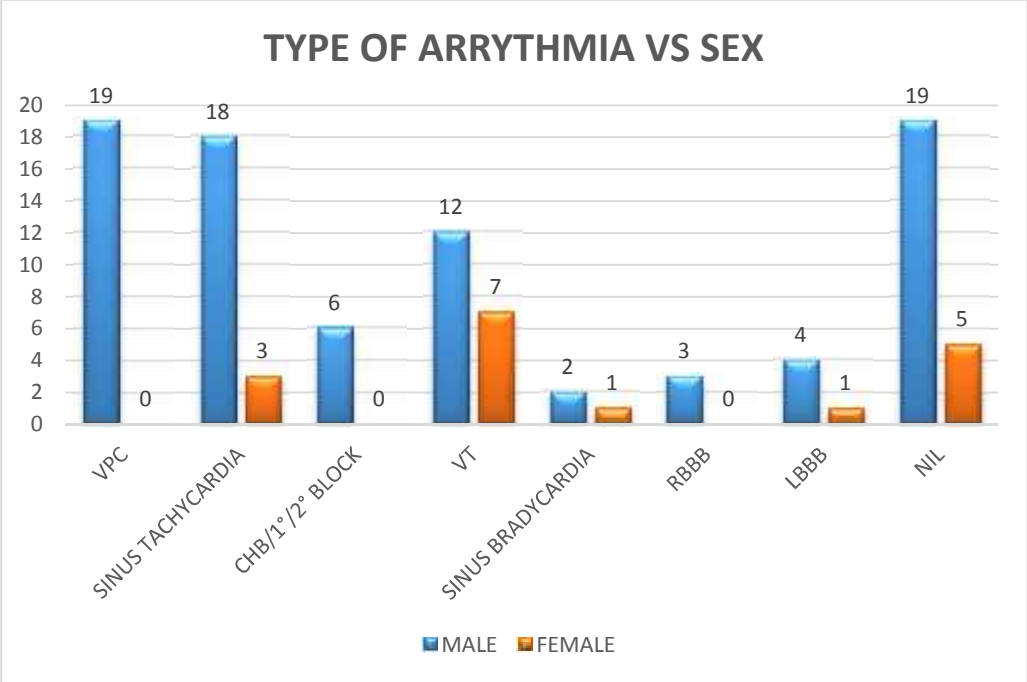
COMMON TYPE OF ARRYTHMIA IN AGE GROUP

AGE (IN YEARS)	TOTAL NO OF PTS	ARRTHYMIA PRESENT	COMMON TYPE
<40	9	4	LBBB(2)
41-50	23	17	VT(5)/VPC(5)
51-60	31	26	VPC(9)/VT(7)
61-70	25	18	ST(7)/VT(5)
>70	12	11	ST(5)

TYPE OF ARRYTHMIA BASED ON SEX

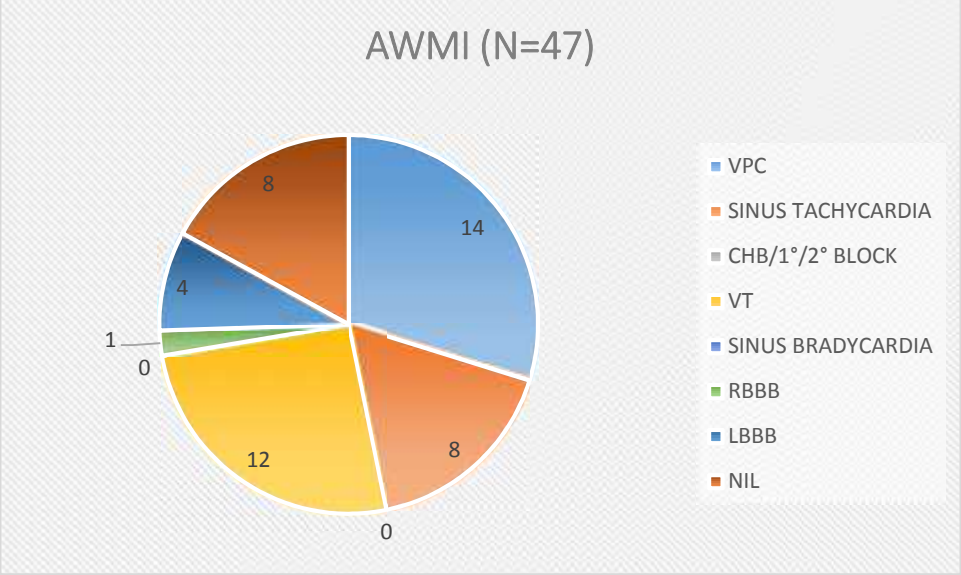
TYPE OF ARRYTHMIA	SEX	
	MALE	FEMALE
VPC	19	0
SINUS TACHYCARDIA	18	3
CHB/1°/2° BLOCK	6	0
VT	12	7
SINUS BRADYCARDIA	2	1
RBBB	3	0
LBBB	4	1
NIL	19	5

AMONG MALE VPC IS MOST COMMON AND AMONG FEMALE VT IS MOST COMMON

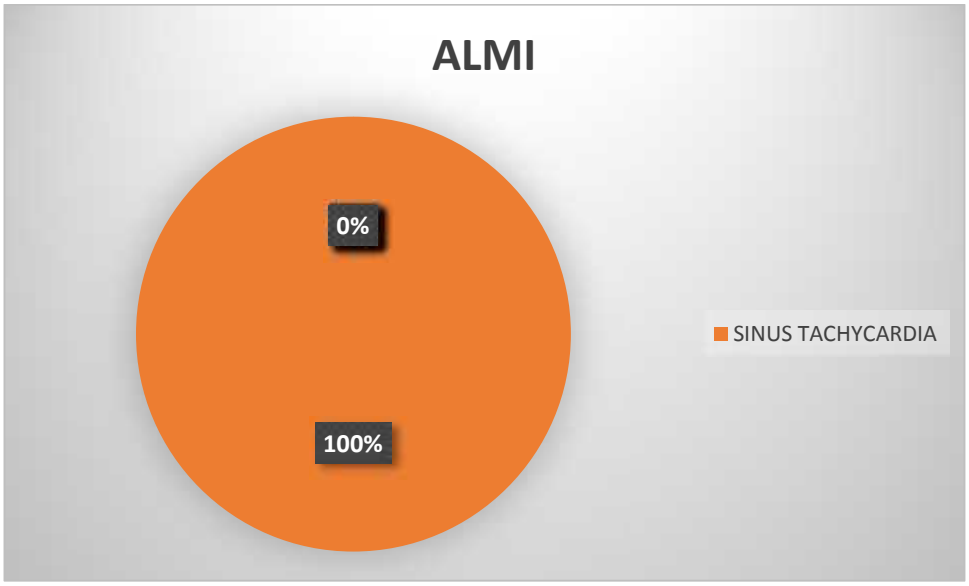


TYPE OF MI	
TYPE OF ARRYTHMIA	**AWMI (N=47)**
VPC	14
SINUS TACHYCARDIA	8
CHB/1°/2° BLOCK	0
VT	12
SINUS BRADYCARDIA	0
RBBB	1
LBBB	4
NIL	8

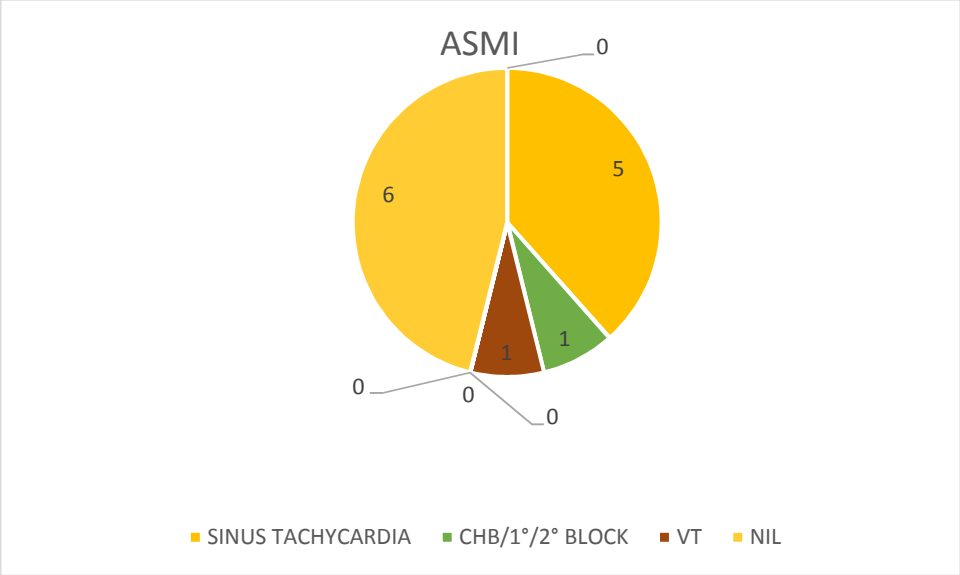
VPC is the most common arrhythmia seen in patients with AWMI.



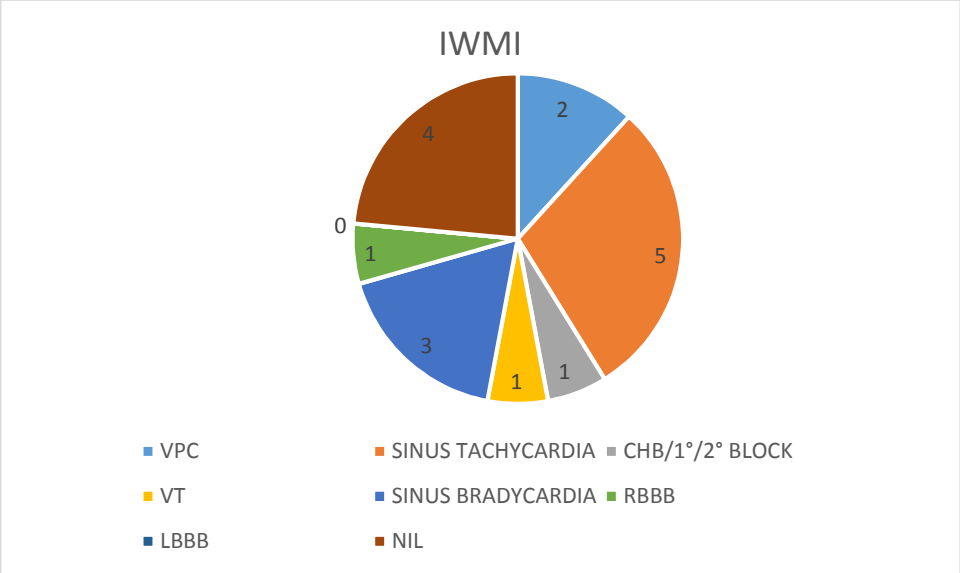
TYPE OF ARRYTHMIA	TYPE OF MI
	ALMI
VPC	0
SINUS TACHYCARDIA	2
CHB/1°/2° BLOCK	0
VT	0
SINUS BRADYCARDIA	0
RBBB	0
LBBB	0
NIL	0



TYPE OF ARRYTHMIA	TYPE OF MI
	ASMI
VPC	0
SINUS TACHYCARDIA	5
CHB/1°/2° BLOCK	1
VT	1
SINUS BRADYCARDIA	0
RBBB	0
LBBB	0
NIL	6

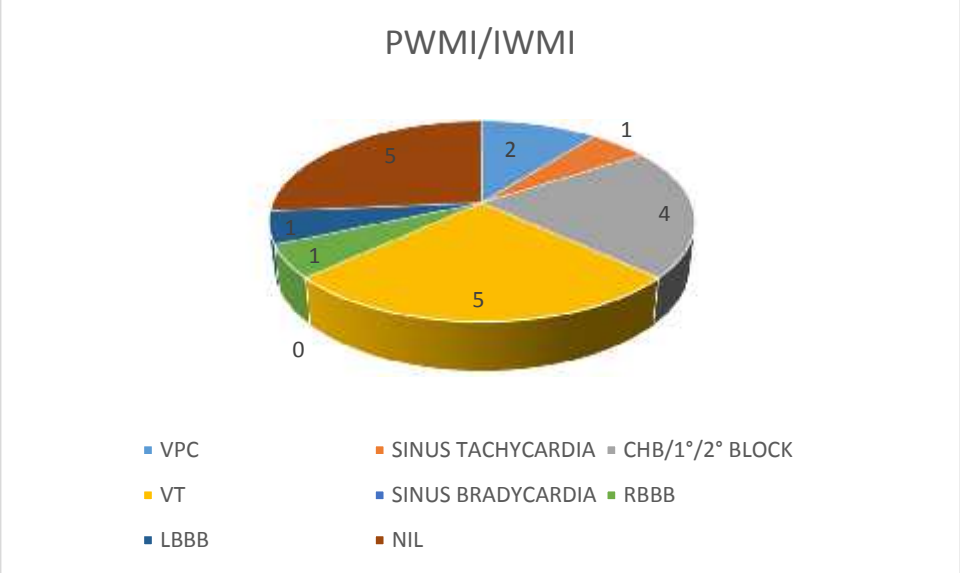


TYPE OF ARRYTHMIA	TYPE OF MI
	IWMI
VPC	2
SINUS TACHYCARDIA	5
CHB/1°/2° BLOCK	1
VT	1
SINUS BRADYCARDIA	3
RBBB	1
LBBB	0
NIL	4

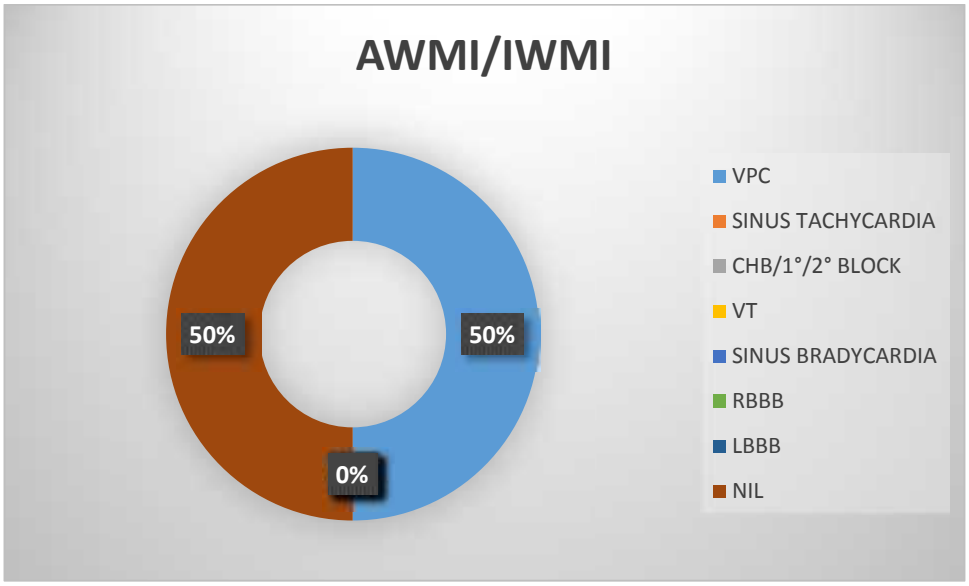


TYPE OF ARRYTHMIA	TYPE OF MI PWMI/IWMI
VPC	2
SINUS TACHYCARDIA	1
CHB/1°/2° BLOCK	4
VT	5
SINUS BRADYCARDIA	0
RBBB	1
LBBB	1
NIL	5

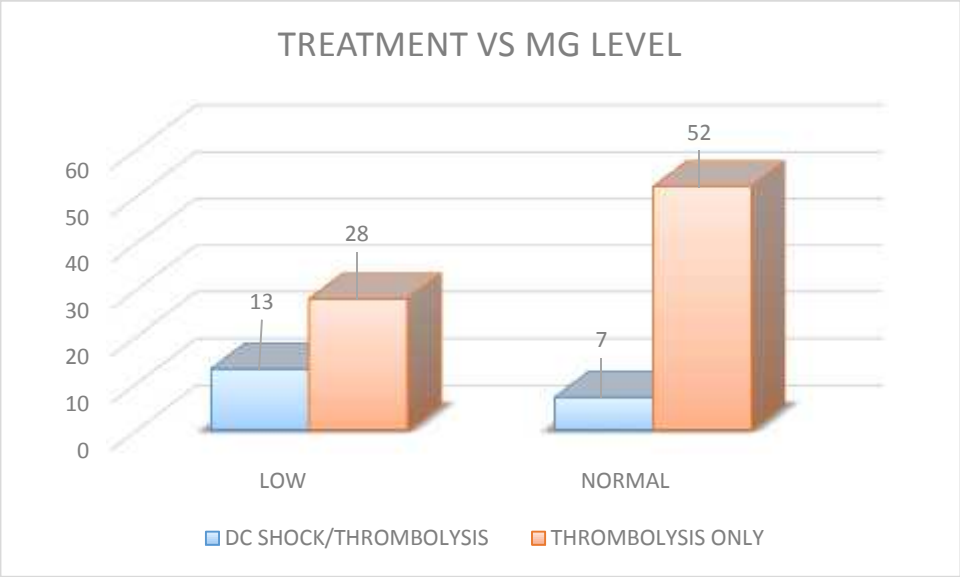
HEART BLOCK IS PREDOMINANTLY SEEN IN PATIENTS WITH INFERIOR WALL AND POSTERIOR WALL MI.



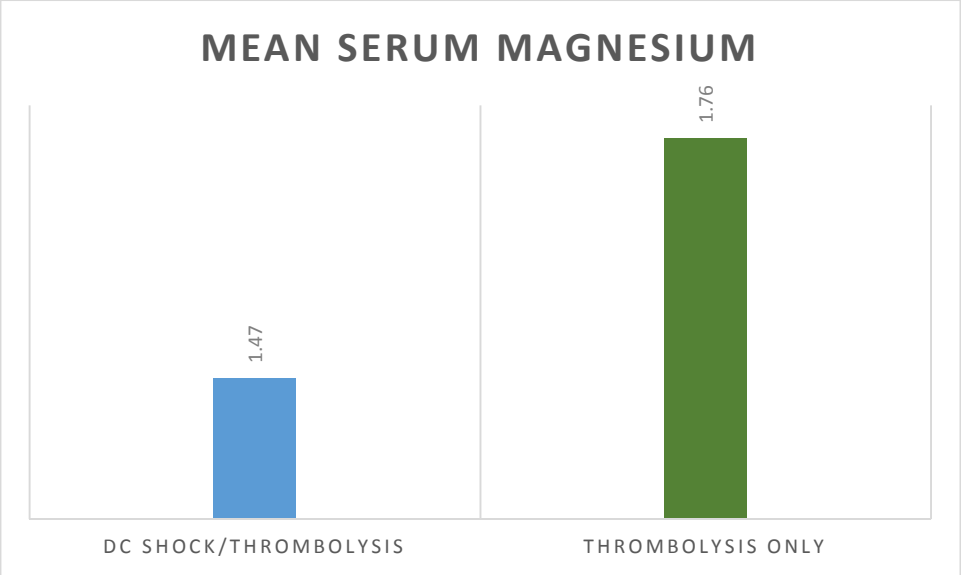
TYPE OF ARRYTHMIA	TYPE OF MI AWMI/IWMI
VPC	1
SINUS TACHYCARDIA	0
CHB/1°/2° BLOCK	0
VT	0
SINUS BRADYCARDIA	0
RBBB	0
LBBB	0
NIL	1



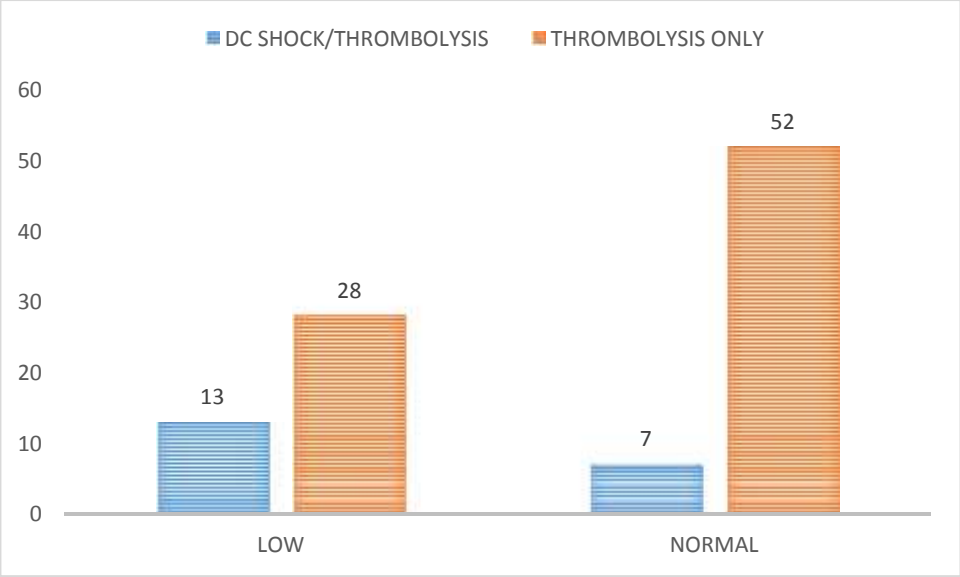
TREATMENT	SERUM MAGNESIUM	
	LOW	NORMAL
DC SHOCK/THROMBOLYSIS	13	7
THROMBOLYSIS ONLY	28	52
CHI SQUARE TEST		
P VALUE-0.015		
ODDS RATIO- 3.4		
SIGNIFICANT		



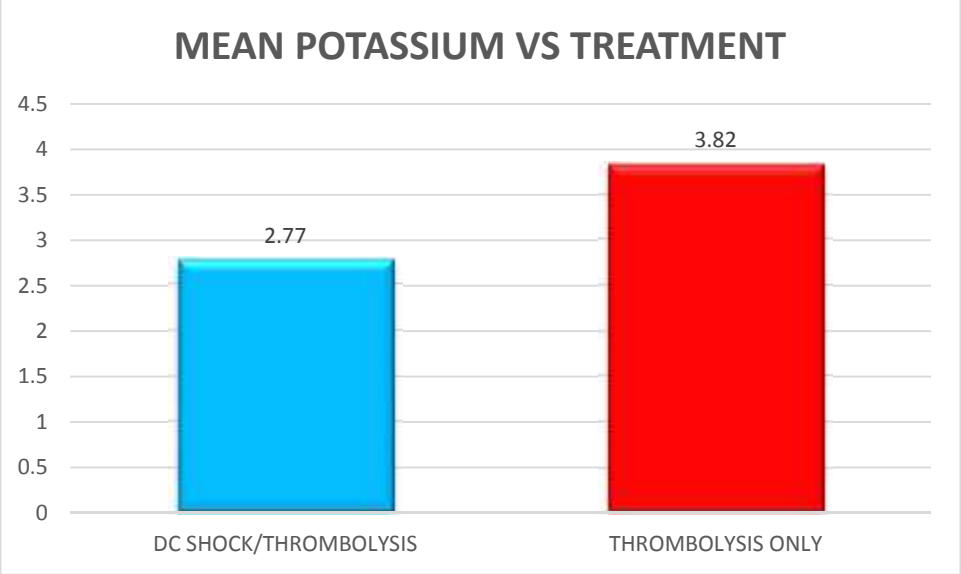
TREATMENT	SERUM MAGNESIUM	
	MEAN	STANDARD DEVIATION
DC SHOCK/THROMBOLYSIS	1.47	0.43
THROMBOLYSIS ONLY	1.76	0.24
UNPAIRED T TEST		
P -0.015		
SIGNIFICANT		



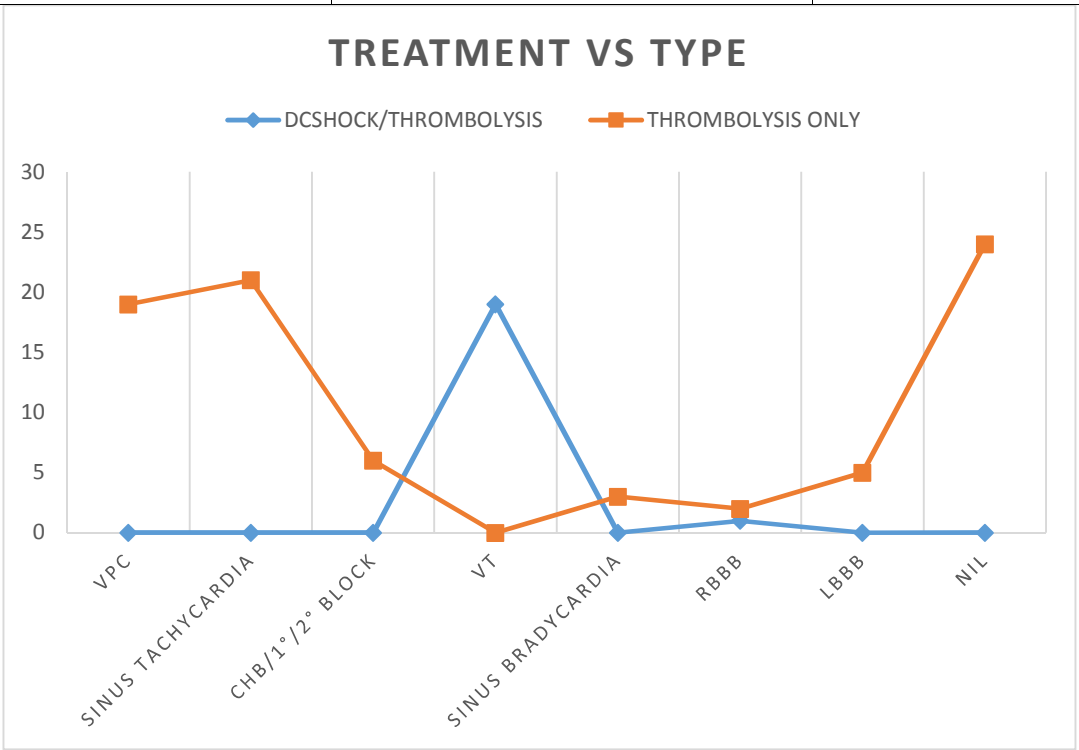
TREATMENT	SERUM POTASSIUM	
	LOW	NORMAL
DC SHOCK/THROMBOLYSIS	13	7
THROMBOLYSIS ONLY	28	52
CHI SQUARE TEST		
P VALUE-0.001		
ODDS RATIO- 15.92		
SIGNIFICANT		



TREATMENT	SERUM POTASSIUM	
	MEAN	STANDARD DEVIATION
DC SHOCK/THROMBOLYSIS	2.77	0.56
THROMBOLYSIS ONLY	3.82	0.48
UNPAIRED T TEST		
P -0.001		
SIGNIFICANT		



TYPE OF ARRYTHMIA	TREATMENT	
	DC SHOCK/THROMBOLYSIS	THROMBOLYSIS ONLY
VPC	0	19
SINUS TACHYCARDIA	0	21
CHB/1°/2° BLOCK	0	6
VT	19	0
SINUS BRADYCARDIA	0	3
RBBB	1	2
LBBB	0	5
NIL	0	24



Out of 100 patients with Acute MI, 41 patients had low serum magnesium levels of which 35 patients developed arrhythmias of which 13 patients developed ventricular tachycardia which was statistically significant with p value 0.008.

DISCUSSION

Strengths and weaknesses of the study design.

Strengths:

Defining the case:

The diagnosis of ST segment elevation Myocardial infarchon is very simple.

Methods: This study needed only serum samples for the estimation of magnesium and potassium which can be estimated relatively easier in central laboratory. Arrhythmias are easily diagnosed by electrocardiogram and continuous cardiac monitoring.

Confounding factors:

The study population is relatively homogenous. No patients had previous history of CAD / Heart failure on Diuretics which would have an effect on the result obtained. And also these patients are not presented with ketoacidosis which would alter serum magnesium values.

The objective nature of the result:

The results obtained are the concentration of serum magnesium and serum potassium in acute MI patients. And arrhythmias are documented. Questionnaires were not required. Thus the result is not influenced by the subjective variation of either the patient or the examiner.

Weaknesses:

Small sample size:

The number of cases included in the study population was 100. The study was done over a period of 18 months. It is highly difficulty to find out a case of STEMI without the frequent co-morbidities like hypertension and diabetes mellitus which also has an impact over the serum magnesium levels. Since the number of sample is 100. It is non-representative of general population.

The internal environment of the patient:

The samples were collected within 6 hours of admission into the hospital. Though the time interval is relatively short, the internal environment in a patient with acute myocardial infarction vary significantly over time due to fluctuating catecholamine levels, thus affecting the magnesium content.

Incidence of Reperfusion Arrhythmias:

Patients with STEMI are treated with fibrinolytic therapy in our hospital. It is associated with varying incidence of reperfusion arrhythmias which does not have a co-relation with magnesium and potassium levels. And they are no definite criteria to establish reperfusion arrhythmias.

PREVIOUS STUDIES

Before discussing about the results obtained a brief review about the results and conclusion of previous studies on this topic of serum magnesium levels in acute myocardial infarction patients.

1. **Serum magnesium in acute myocardial infarction:** Actamedica Scandinavia. Volume 206, Issue 1-6 pages 59-66, December 1980.

   - Thomas Dyckner

During the one and a half years of the study, 342 patients with acute myocardial infarction were treated at Serafimerlasarettet. The acute MI group had significantly lower serum magnesium levels than a reference group. The incidence of Ventricular ectopics, Ventricular tachycardia and Ventricular fibrillation were significantly higher in the hypomagnesaemia patients.

2. **Magnesium and Acute Myocardial infarction. Transient Hypomagnesaemia Not Induced by Renal magnesium Loss.**

Arch Intern Med. 1986; 146(5) :872-874.

H. Sandvad Rasmussen, MD; P. Arup, MD; S. Hojberg, MD Blood and urine samples were taken during the time of admission. Urine samples were taken every 8 hours for the next 7 days. Both urine and blood magnesium were analyzed. 13 patients were found to have MI.11 normal people were taken as controls. The acute myocardial infarction patients had significantly lower levels of serum magnesium. The urine concentration of magnesium did not increase with time. This shows that the hypomagnesaemia is not due to the renal loss magnesium. The mechanism is due to a shift from the extracellular compartment, maybe due to sequestration with the increased levels of free fatty acids.

3. Serum magnesium and potassium in Acute Myocardial Infarction

Arch intern Med. 1987; 147(3): 465- 469

-Henryk Kafka, MD; Lorrie Langevin, RN.

Over a period of 13 months, serum magnesium and potassium levels were measured in 590 patients admitted in a coronary care unit. Hypokalemia occurred in 17% of the patients. However, hypomagnesaemia occurred in ten of the thirteen patients with myocardial infarction and hypomagnesaemia. However the mean levels of serum magnesium levels in the normal healthy population was significantly higher than the reference levels. So the findings in the study may not be applicable to outside population because of higher magnesium content of soil in the selected study area of south eastern Ontario.

In the study conducted in our ICCU, the most common age group with acute MI is 51-60 years (31%). Anterior wall MI (47) is the most common type followed by inferior and posterior wall MI (19). 76 % of the patients developed arrhythmias. The most common type of arrhythmias are Sinus tachycardia (21), ventricular ectopics (19) and ventricular tachycardia (19) followed by bundle branch blocks (8), AV block (6) and sinus Bradycardia (3).

Low serum magnesium levels and low potassium levels was observed in 41% and 38% respectively. The mean serum magnesium levels in sinus tachycardia and ventricular ectopic is 1.77 mg/dl and 1.75 mg/dl respectively. The mean serum magnesium levels in patients with ventricular tachycardia is

1.44 mg/dl and mean serum potassium level is 2.7 meq/l which was statistically significant p value 0.008

Hence it shows that low serum magnesium and potassium levels that occurred in a patient with Acute MI developed life threatening ventricular arrhythmias primarily ventricular tachycardia. This was in accordance with the previous studies that were done in the past.

This opens to widespread discussion that whether prophylactic intravenous magnesium or oral magnesium tablets are helpful in preventing life threatening ventricular arrhythmias. Several clinical trials were conducted on the prophylactic role of magnesium therapy, which are as follows

Intravenous magnesium sulphate in suspected acute myocardial infarction: results of the second Leicester Intravenous Magnesium Intervention Trial (LIMIT-2)

Lancet. 1992 Jun 27; 339(8809):1553-8

Here they conducted a randomized, double blind, placebo controlled study in 2316 patients with suspected acute myocardial infarction who received either intravenous magnesium sulphate (8 mmol over 5 min followed by 65 mmol over 24 h) or physiological saline. The primary outcome measure was 28-day mortality, which was ascertained in 99.3% of patients.

The groups were well balanced for prognostic factors. By intention-to-treat analysis mortality from all causes was 7.8% in the magnesium group and 10.3% in the placebo group (2p = 0.04), a relative reduction of 24% (95% confidence interval 1-43%). Within the coronary care unit the incidence of left ventricular failure was reduced by 25% (7-39%) in the magnesium group (2p = 0.009).

The side-effects of magnesium treatment were transient flushing, related to speed of injection of the loading dose, and an increased incidence of Sinus Bradycardia.

There was a considerable enthusiasm for the routine use of intravenous magnesium in patients with MI, based on the findings of the LIMIT 2 trail which observed a 24 % reduction in mortality compared with placebo. The larger ISIS 4 and MAGIC (magnesium in coronary arteries) failed to duplicate this effect.

However, some have speculated that the lack of effect in ISIS 4 was because of delayed administration or low control group mortality. In the modern era, magnesium is not routinely used other than to replete serum magnesium levels lower than 2.0 mcg/dl or for the management of torsade de pointes.

But this study conducted in our ICCU, Department of Cardiology there is a positive correlation between low serum magnesium levels and ventricular arrhythmias by which we would recommend for the early serum estimation of magnesium and potassium levels and to replete it when low. The preferred dose is 1 to 2 g IV over 5 minutes. It also requires several large scale studies to negate the weaknesses in ISIS 4 and MAGIC trails and to prove the efficacy of prophylactic magnesium therapy.

SUMMARY

1. Most commonly patients with AMI were in the 6th decade.
2. Acute myocardial infarction is most commonly seen in males.
3. AWMI is the most common type of MI followed by IWMI with PWMI.
4. The most common type of arrhythmias in AMI are Sinus tachycardia>ventricular ectopics >ventricular tachycardia.
5. Low serum magnesium levels and low potassium levels was observed in 41% and 38% respectively.
6. The mean serum magnesium levels in patients with ventricular tachycardia are 1.44 mg/dl and mean serum potassium level is 2.7 meq/l which was statistically significant p value 0.008.

   This shows that low magnesium and low potassium occurring in patients with AMI leads to life threatening ventricular arrhythmias.

CONCLUSION

We conclude the magnesium levels do fall significantly in patients with Acute Myocardial Infarction and low serum magnesium in these subset of patients develop life threatening ventricular arrhythmias in the initial 48 hours. Hence we recommend for the early estimation of serum magnesium levels in patients with Acute MI and to replete it when low which would be lifesaving. More such studies are needed especially with a bigger sample size to find out the role and efficacy of prophylactic magnesium therapy.

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Whang R[1], Whang DD, Ryan MP.

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Rude RK[1]

24.Overview of Basic Mechanisms of Cardiac Arrhythmia

Charles Antzelevitch, PhD, FHRS[*] and Alexander Burashnikov, PhD, FHRS

25. Monophasic action potentials in the diagnosis of triggered arrhythmias☆

Douglas P. Zipes

PROFORMA

NAME:

AGE:

SEX:

IP NO:

DATE OF ADMISSION:

DATE OF DISCHARGE:

PRESENTING COMPLAINTS:

CHEST PAIN

PALPITATION

SYNCOPE

DYSPNEOA

SWEATING/GIDDINESS

COMORBIDITIES:

TYPE2DM/SHT/BA/TB/CKD/CVA

VITALS:

BP:

PR:

SP02:

ECG (DAY 1, 2, 3):

CARDIAC BIOMARKERS:

CPK-MB:

SERUM MAGNESIUM:

SERUM POTASSIUM:

LFT:

RFT:

DIAGNOSIS:

TREATMENT FOR ARRYTHMIAS:

DC SHOCK/ANTI ARRYTHMIC DRUGS

ECHO:

CONDITION ON DISCHARGE:

NAME	AGE	SEX	TYPE OF MI	B.P	P.R	ARRYTHMIAS NOTED	SR MAGNESIUM	SR. POTASSIUM	TREATMENT
SIMON RAJA	39	M	STEMI/AWMI	?	?	VT	1.5	2.2	
PETCHIAMMAL	65	F	STEMII/IWMI	?	?	VT	1.3	2.5	DC SHOCK/THROMBOYLSIS
KUMARESAN	53	M	STEMI/IWMI/PWMI	?	?	VT	1.2	1.9	DC SHOCK/THROMBOYLSIS
BOOTHAPANDI	45	M	STEMI/AWMI	?	?	VT	1.9	3.1	DC SHOCK/THROMBOYLSIS
SUDALAIMADI	45	F	STEMI/AWMI	?	?	VT	0.7	2.4	DC SHOCK/THROMBOYLSIS
PERUMAL	70	M	STEMI/AWMI	?	?	VT, RBBB	1.7	2.4	DC SHOCK/THROMBOYLSIS
ARUMUGAM	52	M	STEMI/IWMI/PWMI	?	?	VT	1.6	2.9	DC SHOCK/THROMBOYLSIS

PAPPU	50	F	STEMI/AWMI	?	?	VT	1.7	2.3	DC SHOCK/THROMBOYLSIS
GANAPATHY	50	F	STEMI/AWMI	?	?	VT	0.9	2.9	DC SHOCK/THROMBOYLSIS
KASIPANDI	62	M	STEMI/IWMI/PWMI	?	?	VT	0.6	3.1	DC SHOCK/THROMBOYLSIS
	60	M	STEMI/AWMI	?	?	RBBB,VT	2.1	3.6	DC SHOCK/THROMBOYLSIS
SUSILA	55	F	STEMI/ASMI	?	?	VT	2.3	3.4	DC SHOCK/THROMBOYLSIS
SUNDARAM	55	M	STEMI/AWMI	?	?	VT	1.1	2.5	DC SHOCK/THROMBOYLSIS
LAKSHMI	78	F	STEMI/AWMI	?	?	VT	2.1	3.9	DC SHOCK/THROMBOYLSIS
SHANUMAGA	70	F	STEMI/IWMI/PWMI	?	?	VT	1.3	2.6	DC SHOCK/THROMBOYLSIS
GANESAN	48	M	STEMI/AWMI	?	?	VT,RBBB	1.2	2.3	DC SHOCK/THROMBOYLSIS
MADASAMY	40	M	STEMI/AWMI	120/80	70	LBBB	1.6	3.9	THROMBOLYSIS
SANKARAN	43	M	STEMI/IWMI/PWMI	130/90	70	SINUS BRADYCARDIA,LBBB	1.8	3.7	THROMBOLYSIS
BISMIBEGAM	48	F	STEMI/AWMI	150/100	90	LBBB	2.1	4.1	THROMBOLYSIS
ESAKKIPANDI	50	M	STEMI/EXT AWMI	140/90	90	LBBB	2	3.4	THROMBOLYSIS
GNANASELVAN	50	M	STEMI/IWMI/PWMI	100/70	80	RBBB, CHB	2	3.8	THROMBOLYSIS/ATROPINE
SELVAM	35	M	STEMI/AWMI	200/120	86	LBBB	1.4	3.2	THROMBOLYSIS
CHELLAPA	67	M	STEMI/AWMI	110/70	118	VPC,ST	2.1	3.7	THROMBOLYSIS
SUBBAIAH	65	M	STEMI/AWMI/IWMI	100/70	84	VPC	1.6	3.9	THROMBOLYSIS
SHANKARAN	65	M	STEMI/IWMI/PWMI	90/60	60	VPC	1.5	3.3	THROMBOLYSIS
SHANMUGAIAH	53	M	STEMI/AWMI	110/80	112	VPC, ST	1.7	3.5	THROMBOLYSIS
SUDALI	54	M	STEMI/AWMI	130/80	124	VPC,ST	1.6	3.9	THROMBOLYSIS
RAJENDRAN	49	M	STEMI/AWMI	120/80	90	VPC	2.1	4.2	THROMBOLYSIS
SUDALAI	55	M	STEMI/AWMI	110/60	126	VPC, ST	3	4.6	THROMBOLYSIS
RAMACHANDRAN	55	M	STEMI/AWMI	150/110	80	VPC	1.6	3.1	THROMBOLYSIS
JEYARAM	63	M	STEMI/AWMI	160/100	90	VPC	2.1	3.9	THROMBOLYSIS
ANNAPOORANAM	74	M	STEMI/AWMI	110/70	89	VPC	1.7	3.7	THROMBOLYSIS
SUNDARAM	55	M	STEMI/IWMI/PWMI	100/80	128	VPC,ST	1.8	4.1	THROMBOLYSIS
THANGARAJ	56	M	STEMI/IWMI	100/70	90	VPC	1.5	3.1	THROMBOLYSIS
MUTHUPANDI	51	M	STEMI/IWMI	90/60	50	VPC	1.9	3	THROMBOLYSIS
JEYARAM	60	M	STEMI/AWMI	180/100	96	VPC	1.4	2.9	THROMBOLYSIS
SUDALAI	48	M	STEMI/AWMI	130/80	88	VPC	1.8	3.8	THROMBOLYSIS
ESAKKI	45	M	STEMI/AWM	140/90	90	VPC	1.9	4.1	THROMBOLYSIS

RANGARAJ	45	M	STEMI/AWMI	100/70	120	VPC, ST	1.8	4.9	THROMBOLYSIS
CHELLAPA	67	M	STEMI/IWMI	90/60	70	NIL	1.5	3.2	THROMBOLYSIS
MANI	45	M	STEMI/AWMI	100/70	89	NIL	1.7	3.8	THROMBOLYSIS
CHELLAIYA	51	M	STEMI/IWMI	120/80	90	NIL	1.8	4.1	THROMBOLYSIS
RAMAMMAL	73	F	STEMI/ASMI	120/70	88	NIL	1.4	3	THROMBOLYSIS
SURESH	39	M	STEMI/ASMI	130/100	80	NIL	1.8	4	THROMBOLYSIS
SUNDARAM	43	M	STEMI/AWMI	150/100	95	NIL	1.7	4.3	THROMBOLYSIS
MANIKKAM	60	M	STEMI/AWMI	100/70	90	NIL	1.6	4.6	THROMBOLYSIS
SEYADRAHIYA	65	F	IWMI/PWMI	90/60	50	NIL	1.8	4.4	THROMBOLYSIS
KASTURI	66	F	STEMI/AWMI	100/70	78	NIL	1.5	3.4	THROMBOLYSIS
ESAKKIMUTHU	61	M	STEMI/ASMI	120/80	88	NIL	1.9	4.2	THROMBOLYSIS
MADASAMY	26	m	STEMI/ASMI	120/70	90	NIL	2.2	4.3	THROMBOLYSIS
MARIAPPAN	37	M	STEMI/AWMI	140/90	98	NIL	2	3.9	THROMBOLYSIS
MUHAMMED	50	M	STEMI/AWMI	130/80	80	NIL	2.3	4.5	THROMBOLYSIS
MEENAKSHI	47	F	STEMI/ASMI	140/80	80	NIL	2.3	4.8	THROMBOLYSIS
GANAPATHY	66	M	STEMI/IPWMI	80/50	60	NIL	2	4.3	THROMBOLYSIS
PAZHAIMUTHU	60	M	STEMI/AWMI/IWMI	90/60	90	NIL	1.8	3.8	THROMBOLYSIS
LAKSHMANAN	50	M	STEMI/ASMI	100/70	80	NIL	1.9	3.6	THROMBOLYSIS
MEENAKSHI	70	F	STEMI/IWMI	90/60	90	NIL	1.9	4.1	THROMBOLYSIS
ANBALAGAN	32	M	STEMI/IPWMI	100/70	90	NIL	1.8	3.8	THROMBOLYSIS
MADASAMY	50	M	STEMI/IWMI/LWMI	80/60	56	NIL	1.8	3.7	THROMBOLYSIS
NALLAKNU	34	M	STEMI/IWMI/PWMI	100/60	80	NIL	1.6	3.4	THROMBOLYSIS
PREMKUMAR	55	M	STEMI/AWMI	120/80	79	NIL	1.8	4.2	THROMBOLYSIS
RAJAKANI	60	M	STEMI/EXT AWMI	110/70	94	NIL	2.1	4.3	THROMBOLYSIS
ESWARAVEL	70	M	STEMI/IWMI/PWMI	90/70	66	NIL	1.4	3.7	THROMBOLYSIS
SANKARAN	55	M	STEMI/AWMI	?	?	VT	1.3	2.6	DC SHOCK/THROMBOYLSIS
AMALA	67	M	IWMI/PWMI	?	?	VT	1.2	2.5	DC SHOCK/THROMBOYLSIS
GANESAN	55	M	STEMI/AWMI	?	?	VT	2.3	3.9	DC SHOCK/THROMBOYLSIS
HASSEN MYDEEEN	77	M	STEMI/AWMI	?	?	VT	1.5	2.4	DC SHOCK/THROMBOYLSIS
PONNAIAH	75	M	STEMI/ASMI	100/70	87	I DEGREE HEART BLOCK	1.8	4.7	THROMBOLYSIS
SHANMUGAVEL	70	M	STEMI/IWMI/PWMI	100/60	80	2 DEGREE HEART BLOCK	1.8	4.3	THROMBOLYSIS
MOORTHY	49	M	STEMI/AWMI	100/60	85	VPC, I DEG HB	1.4	3.1	THROMBOLYSIS
GANESAN	55	M	STEMI/IWMI	90/50	55	SB, I DEG HB	1.7	4.2	THROMBOLYSIS
SHIEK MOHAIDEEN	57	M	STEMI/EXT AWMI	110/70	90	VPC, I DEG HB	1.5	3.5	THROMBOLYSIS

THNGASAMY	70	M	STEMI/IWMI/PWMI	80/50	40	CHB	1.3	4	THROMBOLYSIS
AMIRTHARAJ	80	M	STEMI/IWMI/RVMI	80/50	42	RBBB, CHB	1.5	4.3	THROMBOLYSIS
SETHURANI	50	F	STEMI/IWMI	90/60	50	SB, I DEG HB	1.6	4	THROMBOLYSIS
MAHESHWARAN	78	M	STEMI/ILMI	100/70	54	2 DEGREE HEART BLOCK	1.5	3.5	THROMBOLYSIS
SAMY DHAS	51	M	STEMI/IWMI	90/60	52	SB	1.5	3.2	THROMBOLYSIS
JAMAL MYDEEN	36	M	STEMI/IWMI/PWMI	90/50	50	SB,CHB	1.8	4.1	THROMBOLYSIS
SHEIK MYDEEN	51	M	STEMI/IWMI/PWMI	80/50	44	CHB	1.6	3.3	THROMBOLYSIS
KANDASMAMY	52	M	STEMI/AWMI	100/70	110	ST	1.5	3.2	THROMBOLYSIS
RAMACHANDRAN	70	M	STEMI/ASMI	110/70	120	ST	1.7	3.1	THROMBOLYSIS
ASHAN MYDEEN	65	M	STEMI/ALMI	120/80	112	ST	1.5	3.2	THROMBOLYSIS
SORNAMMAL	75	F	STEMI/IWMI/PWMI	110/70	110	ST	1.6	3.4	THROMBOLYSIS
MAHADEVI	55	F	STEMI/ASMI	100/70	106	ST	1.9	3.9	THROMBOLYSIS
SATHAR	60	M	STEMI/AWMI	110/70	110	ST	1.9	4.1	THROMBOLYSIS
RATHINAM	55	M	STEMI/IWMI	100/70	108	ST	2.1	4.3	THROMBOLYSIS
MOHANRAJ	64	M	STEMI/AWMI	110/70	116	ST	1.5	3.2	THROMBOLYSIS
PAULRAJ	74	M	STEMI/IWMI	100/70	114	ST	1.5	3.1	THROMBOLYSIS
VADIVEL	60	M	STEMI/ASMI	110/70	106	ST	1.9	4	THROMBOLYSIS
SAKTHIVEL	71	M	STEMI/ASMI	120/80	126	ST	2.1	4.6	THROMBOLYSIS
RASAIAH	65	M	STEMI/AWMI	110/70	120	ST	1.8	3.9	THROMBOLYSIS
RAJA	58	M	STEMI/IWMI	90/60	112	ST	1.7	3.9	THROMBOLYSIS
ARUNACHALAM	62	M	STEMI/AWMI	110/70	116	ST	1.7	4	THROMBOLYSIS
MARIAPPAN	50	M	STEMI/IWMI	100/60	102	ST	1.8	4	THROMBOLYSIS
SHANMUGAVEL	46	M	STEMI/AWMI	120/80	112	ST	1.9	4	THROMBOLYSIS
MAHALINGAM	70	M	STEMI/ALMI	130/80	140	ST	1.5	3	THROMBOLYSIS
DEVANAYAGAM	52	M	STEMI/AWMI	120/70	124	ST	1.8	4	THROMBOLYSIS
MARIAPPAN	70	M	STEMI/IWMI	100/60	120	ST	2	4	THROMBOLYSIS
GURUSAMY	75	M	STEMI/AWMI	120/80	112	ST	1.7	3.7	THROMBOLYSIS
ESAKKIYAMMAL	75	F	STEMI/ASMI	100/60	124	ST	3.1	4.3	THROMBOLYSIS

COMORBIDITIES

SHT
SHT
TYPE2DM/SHT
SHT
TYPE 2 DM/SHT
TYPE 2 DM
TYPE 2 DM

TYPE 2 DM
TYPE 2 DM
SHT
SHT/TYPE2 DM
SHT/TYPE 2 DM
SHT/TYPE 2 DM
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TYPE 2 DM
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TYPE2DM/SHT
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TYPE 2DM/SHT
SHT

ABBREVIATIONS

STEMI: ST segment Elevation Myocardial Infarction

IHD: Ischemic Heart Disease

AMI: Acute Myocardial Infarction

UA: Unstable Angina

ECG: Electrocardiogram

CK: Creatinine Kinase

rPA: Reteplase

tPA: tissue Plasminogen Activator

CABG: Coronary Artery Bypass Graft

PCI: Percutaneous Coronary Intervention

ACE: Angiotensin Converting Enzyme

RCA: Right Coronary Artery

AIVR: Accelerated Idioventricular Rhythm

RAAS: Renin Angiotensin Activating System

EAD: Early After Depolarisation

DAD: Delayed After Depolarisation

ISE: Ion Sensitive Electrode

AWMI: Extensive Anterior Wall Myocardial Infarction

ALMI: Antero Lateral Myocardial Infarction

ASMI: Antero Septal Myocardial Infarction

IWMI/PWMI: Infero Posterior Wall Myocardial Infarction

VPC: Ventricular Premature Complex

VT: Ventricular Tachycardia

RBBB: Right Bundle Branch Block

LBBB: Left Bundle Branch Block

CHB: Complete Heart Block

DC: Direct Cardioversion